COMPARATIVE STUDY TO ASSESS THE EFFICACY OF EPIDURAL DEXMEDETOMIDINE AND INTRAVENOUS DEXMEDETOMIDINE IN PATIENTS UNDERGOING LOWER ABDOMINAL SURGERIES UNDER EPIDURAL ANAESTHESIA

DISSERTATION SUBMITTED FOR THE DEGREE OF

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BONAFIDE CERTIFICATE

This is to certify that this dissertation titled **"COMPARATIVE STUDY TO ASSESS THE EFFICACY OF EPIDURAL DEXMEDETOMIDINE AND INTRAVENOUS DEXMEDETOMIDINE IN PATIENTS UNDERGOING LOWER ABDOMINAL SURGERIES UNDER EPIDURAL ANAESTHESIA"** is a bonafide record work done by **Dr.S.SIVAKUMAR** under my direct supervision and guidance, submitted to The Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of University regulation for MD, Branch X-Anaesthesiology.

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DECLARATION

I **Dr.S.SIVAKUMAR** solemnly declare that this dissertation titled **"COMPARATIVE STUDY TO ASSESS THE EFFICACY OF EPIDURAL DEXMEDETOMIDINE AND INTRAVENOUS DEXMEDTOMIDINE IN PATIENTS UNDERGOING LOWER ABDOMINAL SURGERIES UNDER REGIONAL ANESTHESIA"** has been done by me. I also declare that this bonafide work or a part of this work was not submitted by me or any other for any award, degree, diploma to any other University or board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfillment of the rules and regulation for the award of Doctor of Medicine degree Branch –X (Anaesthesiology) to be held in April 2013.

Place: Madurai
Date: **Dr.SIVAKUMAR.S**

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INTRODUCTION

Epidural anaesthesia requires larger amounts of local anaesthetic than a spinal anaesthetic. Close attention to the total dose is required to avoid toxicity. Epidural anaesthesia is versatile and can be administered by a single injection or through a catheter. The use of a catheter allows the anaesthesia provider to add local anaesthetics as surgery progresses, extending duration beyond the original dose.

Epidural anaesthesia provides excellent operating conditions for surgical procedures below the umbilicus like inguinal hernia repair,incisional hernia repair,genitourinary procedure and lower extremities surgeries

In lower abdominal surgeries various anaesthetic techniques have improved drastically over last few decades.Manytechniques anddrug regimenswith great success have been tried from time to time to calm the patient and to eliminate the anxiety.

Theprolonged duration of surgeries,sensory and motor block, supine position for a long time and the immobility brings a feeling of discomfort and anxiety to the patients during regional anaesthesia.

Sedatives are given to alleviate the anxiety,but higher doses may produce respiratory depression in some patients. To overcome this disadvantage newer drugslike alpha2Aselective agonist has beentried

toinduce the natural arousable sleep without causing respiratory depression.It has been mixed with local anaesthetic agent as adjuvant.

Dexmedetomidine is a highly selective alpha2 agonist with a greater affinity towards adrenergic receptor than clonidine.The analgesic property and the local anaesthetic property are due to the hyperpolarisation of nerve tissues by altering transmembrane potential and ion conductance at locus ceruleus in the brain stem.The stable hemodynamics and the decreased oxygen demand due to enhanced sympathoadrenal stability, make it apreferable adjuvants with local anaesthetics.It provides better sedation throughout the procedure without causing respiratory depression. The administration of dexmedetomidine through various routes created the interest of this study to compare the routes of administration of the drug to assess the efficacy of basic qualities of the drug like analgesia,sedation, cardiovascular and respiratory responses.

AIM OF THE STUDY

To assess the efficacy of epidural dexmedetomidine and Intravenous dexmedetomidine in patients undergoing lower abdominal surgeries under epidural anaesthesia.

Efficacy is assessed in terms of

I. Sensory block.

II. Motor block.

III. Time for two segment level regression.

IV. Time for rescue analgesia.

V. Intra operative sedation score.

VI. Hemodynamic stability.

HISTORY

WILLIAM STEART HALSTESD (1852-1922) and **RICHARD JOHN HALL (1856-1897)** were the true progenitors of conduction anaesthesia. In 1855, **FRIEDRICH (1828-1890**) became the first person to isolate cocaine, In 1884,**KARL KOLLER (1857-1944)**studied the local anaesthetic effect of cocaine by instilling cocaine into his own eye.

JAMES LEONARD CORNING (1855-1923)was the first to use epidural analgesia in1885. Caudalepidural analgesia has been practised as technique since 1901 by **SICARD** and **CATHELIN**(Paris,France). It was introduced as caudal approach.Lumbar approach for epidural analgesia was tried by **TUFFIER** in 1901.Continous epidural anaesthesia through caudal route was initially described by **EUGEN BOGDAN ABREUL(1899-1975).**

Twenty years later, in 1921, **FIDEL PAGES (Spain,1886-1923)** describedthe injection of anaesthetics into the epidural space in the lumbar and thoracic regions, and this markedly increased the possibilities of the epidural block.**FIDEL PAGES**created interest in midline easy approach for lumbar epidural.

ACHILE MARIO DOGLIOTTI (Italy,1897-1966) was a surgeon.He advocated the use of epidural anaesthesia for a wide variety of procedures since 1931.

DOGLIOTTIfirst described the loss of resistance technique and classic grip of needle and syringe .

VINCENT RUIZ AND ALBERTO GUTIERREZ (Buenos Aires,Argentina)began their practice of epidural anaesthesia based on the work of both **PAGES AND DOGLIOTTI**in 1932.**GUTIERREZ** developed the 'HANGING DROP' sign used to identify the epidural space. In 1941, **ROBERT ANDREW HINGSON** and **WALDO B.EDWARD** developed continuous caudal anaesthesia using indwelling needle.

In 1947, **MANUEL MARTINEZ CURBELO**first described the placement of a lumbar epidural catheter. **JANES** (1926) noted Epidural needles with lateral opening are better suited to avoid dural puncture by epidural pressures exerted during procedure than conventional blunt bevelled needle.

Tuohy needle initially designed for continuous subarachnoid analgesia by **TUOHY**(1945).It was adopted for epidural use by **CURBELO**.

Tuohy needle are produced with a blunted upper rim to avoid shear of the catheter introduced by **LUND (1966).**Later developed epidural needle, catheter and better understanding the concepts of newer drugs pharmacokinetics and pharmacodynamics contributed to the safer epidural anaesthesia.Nowadays, epidural analgesia is an essential technique in anaesthesiology.

The first report on opioids for intra thecal anaesthesia was published in 1905 and on epidural morphine in 1979. Various non opioid adjuvant usedin neuraxial analgesia to improve the efficacy included NMDA antagonists -ketamine , magnesium, GABA agonists-Midazolam, COX inhibitors-ketorolac,Acetyl choline esterase-Neostgmine,Adrenergic agonists-Adrenaline, clonidine.

Alpha 2-adrenoceptor agonists are being increasingly used in anaesthesia and critical care, as they not only decrease sympathetic tone and attenuate the stress responses to anaesthesia and surgery but also produce sedation and analgesia.They are also used as an adjuvant during regional anaesthesia.Dexmedetomidine is the agent in this group approved by FDA in 1999 for use in humans for analgesia and sedation.Dexmedetomidineproduces apowerful antinociceptive effect, mediated at spinal level and systemic redistribution of the drugs lead to hypnotic state.

ANATOMY

Identification of spinous process corresponds to certain landmarks which are essential to identify the appropriate levels to block corresponding dermatomes.

C7-Prominent cervical spine.

T3-Tip of spinous process scapula.

T5-Lowest point in thoracic curve on lying supine.

T7-Tip of inferior angle of scapula.

L3-Highest point in lumbar curve on lying supine.

L4-Highest point of iliac crest.

S2-Posterior superior iliac spine.

Vertebral column:

Cervical and lumbar curvatures are convex anteriorly, thoracic and sacral curvatures are convex posteriorly. In supine position the highest point of the cervical curvatures is at C5 and the lumbar curvature is at L5.The lowest point of the thoracic curvature isat T5, the sacral curvature is at S2.These curvatures have the influence in spreading of local anaesthetic in the subarchnoid space.

The typical lumbar vertebra has kidney shaped body and a triangular vertebral foramen .The spine is straight when viewed from back and square shaped when viewed from sideways.There are noarticular facet for ribs as in thoracic vertebra. The stability and elasticityof vertebral column is provided by various ligaments. Supra spinous ligament connects as a fibrous cord from C7 spinous process to sacrum. Interspinous ligament is present between the ligamentumflavum anteriorly and the supraspinous ligament posteriorly. Both of the ligaments are thickest and broadest atthe lumbar region.

Ligamentumflavum is a yellow elastic ligament connecting the caudal edge of upper vertebral laminae and cephalad edge of lower vertebral laminae.

Anterior andposterior longitudinal ligaments binds with the vertebral bodies. Intervertebral foramina which transmits the spinal nerves is bounded anteriorly by intervertebral disc, posteriorly by the articulating process, superiorly and inferiorly by the pedicles of adjoining vertebra.

SPINAL CORD AND NERVE ROOTS:

The spinal cord is 42-45 cm in length and occupies the two third of the vertebral canal.

It is a continuation of medulla oblongata and ends as conusmedullaris. The filumterminale stretches downward and attaches to the coccyx.

At birth the spinal cord ends at the lower level of L3. In adult the spinal cord ends atthe level of lower border of L1 or the upper border of L2.In cross section there is an anterior median fissure and a posterior median septum. The anterior median fissure extends 3mm into the cord and contain blood vessels and also where the local anaesthetic solution enters the cord. The posterior sulcuscontain neuroglia.

There are 31 pairs of spinal nerves.The anterior and posterior root cross the subarachnoid space,dura, extradural space and unite in the intervertebral foramen and forms the spinal nerve trunk.Thetrunk is divided into anterior and posterior primary division.The autonomic fibers are more sensitive whereas the motor fibers are least sensitive. The sympathetic preganglionic axonsarise from intermediate lateral horn at the firstthoracic level and extendsupto the second lumbar vertebra.The nerve roots are larger in cervical and in lumbo sacral region than in thoracic region. Extension of sympathetic block higher level than motor block.

Blood supply:

The spinal cord is supplied by one anterior spinal arterywhich is a branch of the vertebral artery formed at the foramen magnum.It lies on the median fissure.It descends downwards from the foramen magnum to the filumterminale.

The spinal cord is also supplied by a pair of posterior spinal artery which is a branch of posterior inferior cerebellar arteries.Lowerbranches provides blood supply to caudaequina.These posterior vessels also supply the nerve roots which emerges through the intervertebral foramina. Anastomosis between anterior and posterior spinal arteries is not found. Anterior radicular and posterior radicular arteriesvariable in size and number which the posterior spinal arteries.One of the anteriorradicular arteries with branches from intercostal arteries and larger in size forms arteriaraicularis magna or artery of Adamkiecz .It provides blood supply tothe lower third of spinal cord.

ANATOMY OF EPIDURAL SPACE :

The epidural space extends from the foramen magnum to the sacral hiatus This space is limited superiorly, by the fusion of the spinal and periosteal layers of the duramaterat the foramen magnum.Inferiorly, it extend upto the sacrococcygeal membrane.

The other boundries of the epidural space include

i. Anteriorly,theposterior longitudinal ligament,vertebral bodies and intervertebral discs.
ii. Posteriorly, the ligamentumflavum,facet joints and laminae.
iii. Laterally, the pedicles and intervertebral foramina.

The epidural space varies with vertebral level ranging from 1.5mm at C5,2.5 mm at T6, 5 to 6 mm at L2 level is the widest epidural space .

Thecontents of the epidural space include nerve roots, fat, areolar tissues,lymphatics,and venous plexus. Epidural space is notvoluminous assub arachnoid space.

The anterior Epidural space is narrow because proximity of dura and anterior surface of vertebral canal. The posterior epidural space is widest occupied by nerves, fat and fibrous tissue.

The local anesthetic spread occur superiorly to the base of skull, inferiorlyto the caudal canal with seepage into anterior sacral foramina and leaks through interveretebral foramina into paravertebral space. Local anaestheticsspreads horizontally through the region of dural cuff with entry into CSFand minor into anterior epidural space.

The rapid entry of local anaesthetic occurs into CSF via arachnoid granulations. The dural space is narrow at the posterolateral aspect of the vertebral canal.

The spread of local anaesthetics injected into epidural space is not predictable due to the fatty areolar tissue that offers resistance as well as the foramina through which the fluid leaks but still this block can be made reliable to provide analgesia.

The space is triangular with the apex in the dorso-medial aspect.Sometimes the dura mater divides the space into two dorsolateral compartments and a ventral compartment. The veins in the space are distended during pregnancy.

PHYSIOLOGICAL CONSIDERATIONS

The epidural anaesthesia results in blockade of autonomic nerves , sensory as well as motor fibres .

Autonomic nervous system:

The preganglionic sympathetic axons are present in the intermedio-lateral horn of spinal cord from first thoracic vertebra to second lumbar vertebra.

They are myelinated and travel along the anterior roots and pass in to the white rami to the paravertebral ganglia.Large concentration of local anaesthetics is required to provide the expected sensory and motor blockade.The motor fibers are less blocked than sensory fibers since motor fibers are covered by duralsheath.The large dose itself cause physiological changes due to increased plasma concentration.Complete sympathetic block lead to unopposed parasympathetic activity and causes bradycardia andhypotension.In epidural anaesthesia hypotension can be controlled by providing titrated dose according to level requirement. Spinal roots and trunk is blocked at axonal level. Peripheral sympathetic block can be achieved by blocking efferent stimulus from T10 to L2 level .

Adrenal medullary sympathetic blockade is achieved by blocking T6 to T11 segments which lead to splanchnic vasodilation and decreased circulating catecholamines.

T1 to T4 segmental block causes cardiac accelerator fibers inhibition and leads to increased parasympathetic activity with bradycardiaupper limb vasodilatation and lower limb compensatory lower limb vasoconstriction.

The particulate size of 0.4-1.5µg cross the dural sheath.Occasionally spread of the drug occurs to the spinal cord itself through the arachnoid villi.Lipid solubility enhances the spread of local anaesthetic whereas the regional blood flow in the nervous tissue is responsible for the rate of removal of local anaesthetic.

Effects on respiratory system:

Sensory afferent pathway blockade reduces nociceptive impulses to respiratory centre and the efferent pathway blockade causes intercostal and abdominal muscle paralysis, it may impair the ability to cough effectively.Epidural analgesia in post operativeperiod provide good pain relief thereby improve the functional residual capacity.Total spinal anaesthesia following inadvertent intradural injection of an epidural local anaesthetics cause higher respiratory centre depression and apnoea as a

consequence of decreased blood supply to medullary centre due to decreased cardiac output.

Indeed spontaneous respiration is a valuable monitor of the adequacy of cerebral blood flow during hypotension. Emphysematous patient breath easily probably due to reflex bronchodialation which results from the effect of hypotension on baroreceptor.

Effects on cardiovascular system:

Higher blockadeis likely to be associated fall in arterial pressure, stroke volume, cardiac output and peripheral resistance.Peripheral venodialation causes pooling of blood and leads to decrease preload to the heart and cause reflex bradycardia.In supine position in pregnancy, inferior vena cava compressed by uterus, causes peripheral venous pooling and engorgement of epidural vessels lead to decreased blood supply to spinal cord. It is promptly treated with lateral positioning,intravenous fluid, vasopressors to maintain mean arterial pressure within normal limit.

Adrenaline absorption from epidural space results in stimulation of the beta receptors and increases the cardiac output but the peripheral resistance decreases and there is a fall in mean arterial pressure.Sympathetic blockade of the lumbar extradural block enhances fibrinolysis above T8 so it is a possible contraindication for prostatectomy.

Liver:

Epidural block up to T5 level with lignocaine alone decreases the hepatic blood flow and decrease the splanchnicvascular resistance.

Effects on Gastrointestinal system:

Epidural block extending from T6 lead to splanchnic sympathetic denervation and results in small contracted gut.Gastric emptying is delayed with epidural morphine but not with epidural local anaesthetics. Epidural local anaesthetics may decrease the incidence of post operativeileus.Intestinal anastomosis performed under epidural analgesia shows lower anastamotic leak incidence.

Thermoregulation and shivering:

Sympathectomy induced vasodialation produce central hypothermia through the convection of heat from central part of body to peripheral tissues.

Incidence of decreasing core body temperature due to loss of heat to environment due to vasodialation below the level of blockade and vasoconstriction and shivering above the level of blockade. Incidence of shivering following epidural block is noticed in one third of the patients.

Epidural anaesthesia may decrease the endocrine and metabolic alteration during surgery by blocking afferent and efferent pathway. The extent of block and surgery site influence the block of nociceptive impulses.

Factors that affect the height of epidural anaesthesia are the following:

Age,Height and Weight of the patient.

Volume ,dose and concentration of the drug.

Position,Gravity, Injection site, Pregnancy and Speed of the injection.

Age

With increase in the age,the local anesthetic requirement is reduced to achieve the same level of blockade as their younger counterpart. The effect of age on epidural blocks have demonstrated a greater spread in older patients.This is thought to be related to a less compliant epidural space and dura mater.but, the clinical effect is usually at most an increase of no more than three to four dermatomes.

Height and Weight:

The correlation between patient height or weight and spread of epidural block is very weak at best and seems to have no clinical significance.

The only instance where it may have an effect is in extremely tall people or in extremely short or in morbidly obese patients.

Gravity

Positioning the patient after injection of local anaesthetic into the epidural space impacts its spread and height, but not to the degree that it does with spinal anaesthesia. A Trendelenburgposition may help in spread of local anesthetics and the reverse Trendlenberg limit its spread.

Volume:

Dosing an epidural can be variable. An epidural should be placed at an appropriate level that corresponds to the dermatome level of the intended surgical procedure since epidurals produce a segmental block.

A small volume of a more concentrated local anaesthetic will produce a very limited but very strong block. The increase in block level is not proportion to the volume increase. Doubling the volume will not double the block spread. It is non linear relationship and doubling the volume will only increase the level about one third to half of the original segment. Cervical and thoracic doses are 0.7 to 1 ml per segment with an initial volume of 10ml. Lumbar level dosesare 1.25 to 1.5 ml per segment.

This variation is due to narrowing of the spinal canal as it progress cranially.Since the dose may be variable from patient to patient, it is

important to give the epiduraldose in increments while continually assessing block progression. A segmental block for epidural analgesia would require a smaller dose.The dose of local anaesthetics administered in the thoracic region should be decreased by 30-50% due to a decrease in compliance and volume.

Concentration:

Concentrationof the drug is relatively unimportant in determining block spread.If drug concentration is held constant, increasing the volume of local anaestheticsresults in significantly greater average spread.

The concentration of the local anaesthetic generally affects the density of the block, not the spread.Dose & volume, however, are important variables in determining both spread and quality of the epidural block obtained.

Position:

The lateral position is the preferred position to optimize spread. But in someindividuals sitting position is preferred due to anatomical advantages. There isno differences in spread ofblock when comparing the two positions.

Injection site:

Unlike spinal anesthesia, epidural anesthesia produces a segmental block that spreads both caudally and cranially.Theinjection site in determining the spread of an epidural block is controversial .The injection site should be in the middle of the range of dermatomes that needs to be anesthetized and closest to the main nerve roots involved.Lumbar local anesthetic injections of 10ml tend to spread caudad to include all the sacral dermatomesLumbar injections of 20ml volumes produce much better quality sacral blocks and can also extend cranially to include the midthoracic levels.

Pregnancy:

Greater spread occurs at term and early in pregnancy**.**There is no significantdifferences in level of spread between pregnant and non-pregnant patients.

Speed of injection:

The rapid injection will increase the level of spread or decrease the time it takes for the block to set.This has never been shown to make any difference in eitherdrugs should be injected slowly to avoid rapid increases in CSF pressure, headache and increased intracranial pressures.Also, incremental bolus compared with slow, steady injection has shown no

difference in level of spread. Local anaesthetic solutions are injecting through the catheter.

This gradual administration of medication slows the rate of onset of the anesthetic level and controls the development of the sympathetic blockade.This is an advantage with an epidural anaesthesia than spinalanaesthesia.The Spinalanaesthesia is All or None phenomenon, whereas the epidural can be brought up gradually, slowing the hypotensive response.

Possible site of local anaesthetic action:

I. Paravertebral nerve roots.
II. Intradural spinal roots.
III. Dorsal and Ventral spinal roots.
IV. Dorsal root ganglia.
V. The Spinal Cord.
VI. The Brain by diffusion.

Initial blockade is probably a result of anesthetic blockade at the spinal roots within the dural sleeves.

The duralsleeves have a proliferation of arachnoid villi and granulations that effectively reduce the thickness of the dura mater facilitating rapid diffusion of the local anaesthetic from the epidural space, through the dura and into the CSF surrounding the nerve roots.

Then the local anesthetic diffuses into the nerve root itself, producing anesthesia to that particular dermatome. Because epidural anaesthesia is diffusion dependent, relatively large volumes of local anaesthetic are needed to achieve a block that spans several dermatome.

The block goes as high or low as the volume injected. It is not like spinal anaesthesia where the complete block occur distal to the site of injection. It is a differential block dependent on the volume and site of injection.

Spread of local anaesthetic drugs within the epidural space:

Local anesthetics when administered in to the epidural space moves in a horizontal and longitudinal direction.

Theoretically, local anesthetic when injected, it can spread up to foramen magnum cranially and to the sacral foramina caudally.

Clinically, the extent of longitudinal spread is dependent on the volume of the drug injected,hence the cephaladspread of the drug can be limited.In the epidural space the spread of the local anaesthetic solution spreads in only four additional dermatomes. Horizontal spread of the local anaesthetic solution occurs through intervertebral foramina, entering the dural cuff. A small amount of local anesthetic sometimes enters the anterior epidural space.

Diffusion into the CSF occurs through arachnoid granules in to the dural cuff.

Onset of blockade:

The onset of an epidural block can usually be detected within 5 minutes in the dermatomes immediately surrounding the injection site.The time to peak effect differs somewhat among different local anesthetics.Shorter acting drugs usually reach their maximum spread in 15-20 minutes.

Longer acting local anesthetics usually reach their maximum spread in 20-25 minutes.Increasing the dose of local anesthetics speeds the onset of both motor and sensory block

Duration of block:

The duration of the Epidural block depends on the local anesthetics itself, dose,Patient age,use of adrenergic agonists and adjuvants.

Local anaesthetics and duration:

Chloroprocaine is shortest acting,Lidocaine&Mepivacaine are intermediate acting local anaesthetics.

Bupivacaine and ropivacaine produce the longest lasting epidural blocks.Bupivacaineis a long acting agent on sensory block than muscle relaxation.

In lower concentrations bupivacaine seems to have a preferential sensory block with minimal motor effect.

Adrenergic agents and duration:

Adrenaline in a concentration of 5 micrograms per ml (1:2,00,000) is added to the epidural local anaesthetic solution.

The dose of lignocaine with adrenaline is 7mg/kg whereas without adrenaline it is 3mg/kg. It has been shown to prolong the blocks oflignocaine and mepivicaine by as much as 80%. Adding adrenaline with bupivacaine and ropivacaine does not significantly prolongs the duration of surgical anaesthesia.

TECHNIQUE OF EPIDURAL BLOCKADE:

Either the sitting or lateral decubitus positions can be used. Emergency equipment,drugs and monitors should be immediately available.The lumbar region is by far the easiest due tothe angle of the spinous processes.

The largerspaces between adjacent spinous processes.Easily identifiable location by using easy to find iliac crests as land mark.Width of epidural space is greatest at this level.

In epidural space, catheter tip is placed between ligamentumflavum and dura.

Structures encountered in midline approach are skin,subcutaneous tissuethen to penetrate supraspinatous ligament,interspinous ligament, increased resistance offered by ligamentumflavum once the tip of the needle entered into the ligamentumflavum.

Within a few millimeter,a sudden loss of resistance is felt and the needle enter into epidural space. The distance of epidural space from skin varies from 3 to 8 cm. Epidural catheter should be in high tensile strength with low coefficient of friction. It should have atraumatic tip, depth marker and radio opacity property.

The epidural anaesthesia is performed with a blunted tip the Tuohy's needle designed to facilitate passage of a catheter into the epidural space.The blunted tip is also designed specially to avoid puncture of the dura and if it comes in contact with the dura, the blunt tip will hopefully just inwardly push the dura without puncturing it.

Local anaesthetic (Lignocaine 1% plain) is injected at T12-L1 interspace and a skin wheal is raised with an injection of 2-3 mlof lignocaine with the 22 gauge intramuscular needle, and in the center of the skin a wheal is raised and then deeper along the planned injection tract, injecting slowly as they penetrate deeper into the subcutaneous tissue.

Firmly the back of the non-dominant hand against the patient's skin,then the epidural needle and eventually the hub is grasped and once the epidural space is found the needle is held between the thumb and index finger of the non-dominant hand as it stays in contact with the patient's back .This stabilizes the needle and prevents any unwanted movement either in or out which is especially critical once the epidural space is found.

The epidural needle is place bevel up and introduced into the skin.It is passed slowly through the supraspinous ligament and seated in the interspinous ligament before the stylet is removed. The needle is placed in the interspinous ligament the stylet is removed, the needle is slowly advanced using the "Loss of Resistance" technique.

The LOR syringe is typically made of glass and is filled with either 3-4cc of air, normal saline.As the syringe or needle combo is advanced, pressure is applied to the plunger of the syringe by "Bouncing" or intermittently applying pressure to the plunger.The pattern is move-bounce until loss of resistance is obtained.The needle combo should only be moved 0.5-1mm at a time and then tested for resistance or loss of resistance.

The syringe and needle combo is advanced by applying pressure to the needle and not the syringe.

As the needle passes through the ligamentumflavum, resistance increases and “pop” of is felt as the ligamentumflavum is pierced and once the tip of the needle is in the epidural Space.

Once the epidural spaceis reached, pass thestylet through the needle to made sure there are no tissue plugs possibly blocking the flow of CSF with an inadvertent dural puncture.Once it is determined that theneedle tipis in theepidural space, a test dose of 3ml of 1.5% of lignocaine with adrenaline.It is essential to question the patient after the injection of test dose.

The aim of the question is to determine whether inadvertent dural puncture or the possibility of injecting directly into the vascular system.If the drug is in intravascular, an increase in heart rate within 30 seconds is noticed.With an intravascular injection,patient may experience “buzzing” sound in the ears, a metallic taste in the mouth or circumoral numbness.If the drug enters into subarachnoid space weakness of the great toe is noticed.

A 16 G or 18 G catheter is threaded up to a point of 5cms added to the skin to reach the epidural space distance.Initially a resistance is felt at the tip of the needle.A slight push may be needed to advance the catheter smoothly.

The catheter should not be pulled out of the the needle once it has been inserted, as the catheter may trap on the needle tip and shear or cut the tip off.

Hanging drop method is an alternative technique to loss of resistance to identify epidural space. In this method a drop of saline is kept at the edge of the epidural needle hub is sucked into needle once tip reaches the epidural space.

Advantages:

Epidural techniques have the advantage of better control of level and sympathetic blockade.

Continous epidural catheter is useful in

I. Regional anaesthesia including abdominal surgeries and lower limb surgeries
II. Obstetric Analgesia.
III. Prolonged postoperative analgesia
IV. Cases of unpredictable duration and chronic pain control.

Contraindications:

Absolute contraindication:

Patient refusal,Infection at puncture site,Raised intracranial tension, Coagulopathy.

Relative contraindication:

Pre-existing spinal cord disease, deformities of spinal cord, sepsis,chronic headache or backache,hypovolemia

SIDE EFFECTS AND COMPLICATIONS:

The complications are attributed to physiologic changes are hypotension,bradycardia,urinary retention,shivering. Thoseattributable to the procedure are dural puncture resulting in post dural puncture headache,inadvertent high level of block due tointrathecal injection of drugs due to migration of catheter into sub arachnoid space.

Vascular puncture may lead to the development of epidural hematoma which leads to cord compression and its sequence.

Patient may also develop local anaesthetic toxicity due to accidental intravascular injection due to catheter migration.Large dose of local anaesthetics required for epidural anaesthesia and the presence of numerous venous plexus in the epidural space increase the likelihood of substantial systemic absorption.

Patient may presented with mild CNS symptoms like restless,slurred speech,tinnitus to loss of conscious, seizure and cardiovascular collapse.

Nerve injury is very rare,but occur after paraesthesia developed during performance of the technique. Other complications include epidural

abscess, meningitis, accidental subdural injection produce unusual block.Characterized by patchy sensory block and unilateral dominance.

Sub dural placement catheter is dangerous because catheter can abruptly pierce the thin arachnoid membrane and enter into subduralspace.

Differentiation between high epidural and total spinal anaesthesia :

Epidural space ends at foramen magnum cranialy, so the cranial nerves are not blocked by epidural anaesthesia. Even with high sensory blocks there are areas of sensation not affected by epidural anaesthesia because they are innervated byafferent fibres in the cranial nerves .

The occulomotor nerve induces miosis after opioid administration so preservation of this response differentiates high epidural anaesthesia from total spinal anaesthesia.

Signs of successful lumbar epidural block:

I. Feel of heaviness or numbness in the lower limb.
II. Sluggishness or absence if knee jerk or ankle jerk.
III. Relaxation of anal sphincter tone by rectal examination suggests sacral block.
IV. Fall in blood pressure
V. Warm lower extremities due to vasodilatation caused by sympatheticblock.

VI. Scaphoid abdomen due relaxation of abdominal muscles and reduced intra abdominal pressure.

VII. No effective cough reflex or the abdominal wall do not stiffen on coughing with high segmental block.

LIGNOCAINE:

In 1905**, Einhorn** introduced the ester local anaesthetic procaine.

In 1943, **Lofgren** introduced lignocaine in sweden it is a protypical amide local anaesthetic. **Gordh**used lignocaine in 1948. Ester local anaesthetics is instability in solution short action, degradation when exposed to high temperature, increased incidence of allergic reaction .

PHARMACOLOGY OF LIGNOCAINE

Physiochemical properties of Lignocaine:

Lignocaine is a synthetic amide-local anaesthetic of intermediate potency and duration. It is 2-Diethylaminoaceto-2’6’-xylitide It is stable,clourless,crystalline solid.Lignocaine HCL readily souble in water.It is less lipid soluble than bupivacaine. It is a weak base. It is not decomposed by boiling,acids or alkalies.

pKa - 7.6-7.8.

Molecular weight -234.34 g/mol.

Mechanism of action :

Local anaesthetics prevent conduction blockade by inhibiting passage of sodium channels in nerve membranes. Blocking the open sodium channels by lignocaine molecule contributes partial to complete inhibition of sodium permeability thereby slowdown the rate of depolarisation so that threshold potential could not be achieved.Thus the action potential is not propagated.

Sodium channel exist in activated open state, inactivated closed state, rested closed during various phase of the action potential.

Lignocaine selectively inhibit the sodium channels in inactivated state, local anaesthetic molecules stabilise these sodium channels and thus prevent their change to rested closed and activated open states in response to nerve impulses.

Other site of action includes voltage dependent potassium ion channels. Voltage sensitivity calcium ion channel (L-type)may also be blocked .

DISTRIBUTION, UPTAKE AND ELIMINATION:

Around 6-8 times the amount of local anaesthetic is required in epidural anaesthesia to produce the same degree of blockade as produced in subarachnoid block.

This is due to the following factors:

Local anaesthetics are lipid soluble and are absorbed into epidural fat. Local anestheticsmust penetrate the dura mater to block the larger mixed nerve roots are found in the epidural space.

Epidural vessels absorbs a significant amount of local anaethetic solution injected . The peak blood concentration occur 10-30 min after a bolus dose.

The local anaesthetics injected into epidural space are get absorbed initially into epidural veins and are diluted in the blood.

The drug enters into pulmonary circulation where the lungs act as a temporary buffer in the view of protecting other organs from the toxic effects of local anaesthetics. The local anaesthetic is redistributed to vessel rich organs,then to the muscles and fat.

Long acting amide local anaesthetics are bound to alpha-1acid glycoprotein, which binds to the local anaesthetics in high affinity and are metabolised in liver and eliminated through renal excretion.

Distribution:

Lignocaine binds to α1 acid glycoprotein(70%), and the volume of distribution is 91 litres.

Triphasic distribution of Lignocaine –

I. Alpha phase (Rapid distribution phase) drug is distributed to highly vascularised region.
II. Second phase Beta phase (slow disappearance phase) drug is distributed to solely equilibrating tissues with t ½ 9.6 minutes.
III. Last is delta phase (slow transformation and excretion phase) where the t½ is 1.6 hours with clearance of 0.95 litre per minutes.

Metabolism and Excretion:

Lignocaine is metabolised in liver into monoethylglycinexylidide followed by hydrolysis of this metabolite to xylidide. Biotransformation of lignocaine in liver includes oxidative N- dealkylation, Ring hydroxylation, cleavage of the amide linkage and conjugation.

The metabolite has prolonged elimination half time. 75% of xylidide excreted in the urine as 4-hydroxyl 2,6 dimethylalanine. Monoethylglycynexylidide has approximately 80% of lignocaine cardiac protective activity. In liver dysfunction and pregnancy induced hypertension elimination half time is prolonged.Metabolism depends on hepatic blood flow.Lignocaine crosses the blood brain barrier presumably by passive diffusion.

Elimination half –life of this agent is typically 1.5-2 hours.Peak blood level of the drug may occur 5 to 30 minutes.

At a concentration of 1 to 4 microgram free bases per milliliter, arooud 60 to 80 percent of drug is protein bound which is determined by plasma concentration of alpha-1 glycoprotein.

Potency:

Lignocaine has rapid onset of action. Myelinated A delta fibres and Non meylinated C fibre are blocked by same concentration of local anaesthetics.Preganglionic B fibres are more readily blocked by local anaesthetics.Lignocaine has rapid onset of action, for conduction blockade small nerves it is 5-10 min and for larger one 10-15 minutes and for I.V lignocaine 1-2 minutes. Topical application onset vary with percentage of available Lignocaine.

The potency of a local anaesthetic is affected by several factors including Hydrogen ion balance, Fibre type size and myelination,Vascular uptake, Proximity to nerve to be blocked, pH of the environment (acidic pH antagonise the local anaesthetic action).

Hypokalemia and Hypercalcemia antagonise the action of lignocaine.

Pharmacodynamics:

Lignocaine inhibits the sodium channel in peripheral nerves. It prevent impulse conduction.It has membrane stabilising property in cardiac tissues.

It prolongs the phase IV depolarisation thereby it depresses the myocardial automaticity, contractility and conduction.

It is clinically used as antiarrythmic agent to depress ectopic foci and also cause bradycardia and hypotension due to vasodialation effect on vascular smooth muscle.

Intravenous lignocaine cause bronchial smooth muscle relaxation and bronchodialation. It prevent reflex bronchoconstriction followimg intubation.It depress the ventilatory response to hypoxia also depress medullary respiratory highercenter.

Lignocaine has CNS excitatory property. It decreases cerebral blood flow thereby reducing intracranial tension following intravenous administration.Also it reduces the MAC value of Inhalation agent.

Indication:

I. Used for local anaesthesia by infiltration .

II. Regional anaesthesia used in spinal anaesthesia, Epidural anaesthesia, Intravenous Regional anaesthesia, Nerves and Plexus block .

III. Treatment and prophylaxis for life threatening ventricular arrhythmias. Lignocaine is class I B anti arrhythmic agent .

IV. Used in Digitalis toxicity and used following resuscitation from cardiac arrest.

V. Supress sympathetic stimulation following intubation.

Contraindication:

I. History of Allergy or hyper sensitivity to Amide type local anaesthetics.

II. Severe degree of sinoatrial ,Atrioventricular or Interventricular block.

III. CNS infection like meningitis, syphilis, polomyelitis, cranial or spinal haemorrhage.

IV. Tuberculosis or metastaic lesions of spinal cord.

V. Patients with severe shock.

VI. Impaired coagulopathy state for regional anaesthesia .

VII. Myastheniagravis

Drug interaction:

Lignocaine should be cautiously used in patients takingantiarrythmic drugs since it potentiate the anti arrhythmic property.Beta antagonists and cimetidine reduces the clearance of inrtavenous lignocaine.

Phenytoin and lignocaine has additive cardiac depressant activity.

Fluoxamine drastically reduces the elimination of lignocaine. It reduces the Minimum alveolar concentration of Inhalation agents. Prolongs the skeletal muscle relaxant action.

Acute severe alcoholic intoxication centrally depress the cardiovascular system thereby reducing the elimination half time.

Dose:Recommended maximum dose as plain lignocaine is 3 mg/ml with adrenaline (1:2,00,000) 7mg/kg. For reflex suppression 1.5mg/kg Inravenously 60 minutes prior to intubation.

Toxicity:

Reactions due to overdose with lignocaine are systemic manifestation.It involve the central nervous system, respiratory system and cardiovascularsystems.Toxicity lead to medullary depression lead to respiratory arrest, tonic and clonic convulsions and cardiovascular collapse.Cicumoral numbness manifests at 4µg/ml of blood ,convulsion occur at 10µg/ml,coma occur at 15µg/ml,respiratory depression occur at 20µg/ml,cardiovascular collapse occur at 25µg/ml of blood.In unconscious patient cardiovascular collapse may be the initial manifestation.

Treatment:

Patency of airway and adequacy of ventilation must be ensured.

For convulsion small incremental dose of Diazepam or an ultra short acting barbiturates like Thiopentone should be given. If the patient is anaesthetised short acting muscle relaxant should be given.

For cardiac toxicity Blood pressure should be maintained with adequate intravenous fluid administration and vasopressors.

Continuous monitoring for early identification of toxicity.

Adrenaline Effects in the Epidural Space and Spinal cord Blood flow:

Adrenaline at a concentration of 5miccrogram/ml is added local anaestheticsto decrease the absorption. In 1:2,00,000 dilution dose of adrenaline will reduce the peak blood concentration level of lignocaine irrespective of route of administration.Benefits of delayed absorption of drug systemically decreases the drug toxicity, increased neuronal uptake,improve quality of analgesia and prolonging the duration of lignocaine.

To prepare 5 microgram/ml concentration of adrenaline the total volume of the lignocaine to be administered is divided into half . The decimal point moved two places to the left of the divded value. This is the amount of adrenaline to be added to the local anaesthetic total volume it yield 1:2,00,000 dilution.

Another method for adding adrenaline to local anaesthetic preparation is followed as 1: 2,00,000 adrenaline concentration is equal to 5µg/ml. 1mg/ml adrenaline (1:1000) is diluted in 10 ml Normal saline loaded syringe now the concentration is 100µg/ml.

The total amount of Lignocaine in ml is multiplied with 5 gives the amount of adrenaline to be added.

Vasoconstriction in the spinal cord cannot explain these decreased venous concentrations because spinal cord blood flow did not change, thus eliminating the possibility of radicular artery or venous plexus constriction.

Adrenaline induced prolonged exposure to local anaesthetic contributes to increased anaesthetic duration. The local anaesthetic peakplasma concentration are lower when adrenaline is added to epidural drugs.

Adrenaline causes increased cardiac output promoting hepatic uptake and increased renal blood flow and elimination. Adrenaline has high volume of distribution due to increased capacitance.

The prolongation of anaesthetic effect can also be explained by an analgesic effect throughadrenergic stimulation at spinal cord.Adrenaline in epidural can produce segmental hypoanalgesia even not mixed with local anaesthetics.

PHARMACOLOGY OF DEXMEDETOMIDINE

Alpha2-agonist areused in anaesthesia and critical care in recent years because it not only act as sympatholytic agent and reduces stress responses to anaesthesia and surgery but also reduces provide good sedation and analgesia.Dexmedetomidinehydrochloride is a selective alpha2-agonist.

It is a 4-((s)-alpha2,3-trimethylbenzyl) imidazole monohydrochloride or 4- [(1r)-1-dimethylphenyl) ethyl] -3h -imidazole hydrochloride.

Dexmedetomidine compared to clonidine is a much more selective alpha2-agonist,alpha2/alpha1selctivity of dexmedetomidine is 1:1620.It has selective alpha2- adrenoreceptoragonism especially 2A subtype. Dexmedetomidine acts on this receptor provides more effective sedative and analgesic even with relatively higher dose without unwanted vascular effect.

It is used as a short term sedative analgesic in Intensive care unit for less than 24 hours.

Reversal drug for its sedative effect is Atipamezole. These properties render dexmedetomidinesuitable agent during perioperative period as premedication,as anaesthetic adjuvants for general and regional anaesthesia,suitable for postoperative sedative and analgesic.

Alpha2 adrenoreceptors are found in peripheral and central nervous systems,platelet,liver,kidney,pancreas,eye and vascular smoothmuscles.

Alpha2-adrenoceptors found in pre synaptic, post synaptic and extra synaptic location . Alpha2- adrenoreceptors are divided into three sub types .The sub type A prominent in central nervous system it is responsible for sedative, sympatholytic, analgesiceffect.Subtype B found in peripheral vasculature.Subtype C found in Central nervous system is responsible for anxiolytic effect.The sub type 2A shows an inhibition effect over the calcium channel in the locus ceruleus of the brain stem but the subtype 2B shows excitatory effect.

Mechanism of action :

In central nervous system presynaptic activation of alpha2A receptor in the locus cerulus inhibits the release of Norepinephrine leads to sedative and hypnotic effect.

Locuscerelus is the site of origin for the descending medullospinal noradrenergic pathway it an important modulator of nociceptive neurotransmission. Stimulationthe receptorinthisarea terminate the propogation of pain signals leading to analgesia. Post synaptic activation of alpha2 receptor resultsin decreased sympathetic activity leads to hypotension and bradycardia.cardio vagal activity due to augmentation of these receptors.

At spinal cord level dexmedetomidine stimulate substantiagelatinosa on dorsallhorn and inhibits peripheral nociceptive reception thereby inhbits the release of substanceP.

In peripheral nerve preventing Norepinephrine release.Stimulation Alpha2 receptor in blood vessels contraction of vascular smooth muscle.

Pharmacodynamics:

Dexmedetomidine induced sleep qualitatively resembles normal sleep. The majority of patients are effectively sedated but they are easily arousablewhich is the unique feature of dexmedetomidine.

It has no direct action on the heart but the cardiovascular changes is biphasic with bolus of 1µg/kg initially cause transient increase in blood pressure and reflex fall in heart rate in healthy individuals.

Stimulation of beta receptor following bolus intravenenous administration causes transient increase in blood pressure initially. But in rate of infusion this response can be attenuated.

Presynaptic receptor stimulation lead to decrease in Norepinephrine release results in fall in blood pressure and pulse rate. These effect can be managed with Atropine, Ephedrine, and adequiate preloading.

The respiratory depression caused by dexmedetomidine is much less than other sedatives. It also decrease salvation, decreased platelet aggregation, inhibit the release of renin, bowel motility is decreased,

intraocular pressure is reduced, increased elimination of water and sodium. Shivering incidence is decreased by reducing temperature threshold by two degree celcius.

Pharmacokinetics:

Dexmedetomidine undergoes glucuronidation and complete hydroxylation and cytochrome P450 metabolism in liver.

It has rapid distribution phase with half life of 6 minutes and elimination half lifeof 2 hours following intravenous administration.It is the active d-isomer of medetomidine. It has volume of distribution of 118 litres. Dexmedetomidine shows linear kinetics with the infusion rate of 0.2-0.7 µg/kg/hour for 24 hours in intensive care unit,the effect appear in5-10 in and reduced in 30 -60 min.

The average protein binding of dexmedetomidine is 94%. Metabolites are excreted in the urine about 95% and in feces 4%.Dose of dexmedetomidine should be reduced in hepatic and renal impairment. It is available as 100µg/ml.Atipamazole is an antagonist of dexmedetomidine act by increasing central norepinephrine.Duration of action is 2 hours.

Uses in Anaesthesia:

Dexmedetomidine used as premedication for its anxiolytic,sedative, analgesic and sympatholytic properties.

Inraoperatively it is used to reduces stress induced sypmathoadrenal responses during intubation as well as extubation.

It potentiates the effects of intra operativeusing anaesthetic agents regardless of route of administration. It is used as sole anaesthetic agent for minor surgery. With higher plasma concentration of dexmedetomidine decreased muscle force dected by mechanomyography.It decreases opioid requirement resulting in more rapid recovery from anaesthesia. Itprovides intense analgesia and reduces the needfor painmedication in PostAnaesthesiaCareUnit

Post operative analgesic requirement reduced by 50% in cardiac patient dexmedetomidine is associated with lower incidence of shivering.

In regional anaesthesia it is used as adjuvants in epidural analgesia to reduce post operative shivering, used with lignocaine in Inravenous regional anaesthesia to improve quality of analgesia withoutcausing side effect.It is a powerful antinociceptive effect,mediated at the spinal level.

Uses in critical care Management:

Dexmedetomidine has been used in the intensive care for its sedative and analgesic properties and does not produce respiratory depression due to its non opioid mechanism of analgesia.The dosage of Dexmedetomidine is loading dose of 1µg/kg over 10mins and maintainence dosage of 0.2 to 0.7µg/kg/hr.

Dexmedetomidine is diluted in 0.9% normal saline.It is not given as bolus dose as it causes paradoxical hypertension.This hypertension due to stimulation ofpost synaptic alpha2B stimulation. It is recommended for infusion up to 24hours only. Ithas been used continuously as infusion in mechanically ventilated patients prior to extuubation, during extubation and in postextubation period.

It iscatiouslyused in patients with bradycardia, atrio ventricular conduction block, ejection fraction less than 30%, hypotensive, hypovolemic.

Advantages are reduces patient awareness of the environment, reduces patient response to external stimuli, facilitates endotracheal tube tolerance and ventilator synchronisation.

MATERIALS AND METHOD

It is a prospective Randomised study conducted in Goverment Rajaji Hospital,Madurai.

The ethical committee's approval was obtained and informed consent was obtained from the patient,who were included in this study.

Inclusion criteria:

I. ASA Iand II patients

II. Age between 35to 65 years.

III. Both sexes

IV. Patients posted for lower abdominal surgeries

Exclusion criteria :

I. History of allergy to local anaesthetics.

II. Previous neurological deficit.

III. Infection at local site

IV. Height less than 150 cm

V. Weight more than 120 kg.

VI. Patents on beta Blockers,Calcium channel blockers.

VII. Patients with conduction block and arrhythmias.

A total of 60 patients were randomnly divided into two groups of 30 each.

Group ED received dexmedetomidine through epidural route and

Group ID received dexmedetomidine through intravenous route .

Inside theoperation theatre, good Intravenous access was secured and the patient was pre loaded with 10 ml/kg of Normal saline.

The monitors included Spo2, ECG and NIBP.The baseline parameters were recorded (Pulse rate,Blood pressure,Spo2,Respiratory rate).

Patient was put in Right lateral position and epidural was performed. With asepsis and antisepsis of the region, L1-L2 space was identified and infiltrated with 1% lignocaine at skin level and using 16G Tuohy'sneedle and LOR syringe epidural space is identified.

The test dose of 1.5% of 3 ml lignocaine with adrenaline is injected to exclude vascular and intra thecal administration.16G catheter threaded into the space and tip of the catheter placed at T11 level. Patient turned to supine position.

TheED group received 2% lignocaine with adrenaline at 5mg/kg (5µg of adrenaline for each ml of lignocaine in(1:2,00,000) dilution) The adjuvant dexmedetomidine was mixed with local anaesthetic in a dose of 1µg/kg and administered through the epidural catheter.

The ID group received 2% lignocaine with adrenaline only through the epidural route. Dexmedetomidine was administered through the intravenous route in a dose of 1µg/Kgwhich was mixed in 100 ml in normal saline and infused over 10 minutes through a seprate dedeicated intravenous line

Following parameters are noted :

a. Time taken for sensory onset T10.
b. Time of onset of complete motor block.
c. Level of maximum sensory block.
d. Time to two segmental regression
e. Duration of surgery.
f. Time for rescue analgesia.
g. Intraoperative sedation score.
h. Hemodynamic parameters, Blood pressure, Pulse rate, Spo2, Respiratory rate.
i. Complications if any.

The bilateral pin prick method was used to assess sensory blockadelevel and Modified Bromage score was used to asses the motor blockade;Score0 for no block ,1for not able to flex the knee ,3 for not able to flex ankle and knee used for motor blockade.

Sedation was assessed with Ramsay sedation score;1 for patient anxious,agitated,restless, 2 for patient is co operative and calm,3 for patient responds to oral commands only, 4 for exhibits brisk response to loud auditory or light glabellar tap, 5 for sluggish response toglabellar or auditory stimulus, 6 for patient exhibit no response .

Vitals monitoring included pulse rate, Blood pressure, Spo2, respiratory rate for every 2,5,10,15,30,45,60,90,120minutes interval .Fall in blood pressure less than 20% from baseline was considered as hypotension and treated with 6 mg ephedrine and fall in pulse rate lessthan 50/min was considered as bradycardia and treated with Inj.Atropine 0.3 mg.

OBSERVATION AND RESULTS

A total of 60 patients undergoing lower abdominal surgeries under epidural anaesthesia is divide into two group contain 30 patients in each group .Epidural anaesthesia (ED) 2% lignocaine 5mg/kg with adrenaline 5 µg/ml with dexmedetomidine 1µ g/kg given in epidural route .Intravenous dexmedetomdine (ID) 2%Lignocaine 5mg/kg with Adrenaline 5µg/ml given through epidural route dexmedetomidine 1µg/kg given in 100 ml Normal saline over10 minutes.

Data analysis was done with the help of computer using Epidemiological Information Package (EPI) developed by centre for Disease control,Atlanta.Using this software range, frequencies, percentages, means, standard deviation, chi square, and 'p'values were calculated .Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variablesand Yate's chi square test for qualitative variables. A 'p' value less than 0.05 is considered as significant relationship and less than 0.0001 as high significant.

Group ED - Epidural Dexmedetomidine

Group ID - Intravenous Dexmedteomidine

PROFILE OF CASES STUDIED

Table 1 : Age

Group	**Age in years**		
	Range	**Mean**	**SD**
Group ED	40-64	53.4	6.2
Group ID	45-65	53.5	5.0
'p'value	0.9705 Not significant		

Mean age of group ED was 53.4 years and group ID was 53.5 years .There was no significant statistical difference.(p=0.9705) .

Table 2 : Sex distribution

Sex	Group ED		Group ID	
	No.	**%**	**No.**	**%**
Male	16	53.3	17	56.7
Female	14	46.7	13	43.3
Total	30	100	30	100
'p'	0.7969 Not significant			

53.3% in **G**roup ED and 56.7% in Group ID were Male. 46.7%in Group ED and 43.3% in Group ID were Female .The sex distribution did not have any statistical significance.

Table 3 :BMI

Group	BMI		
	Range	**Mean**	**SD**
Group ED	20.2 – 27.4	23.2	1.8
Group ID	16.4 – 36.1	24.4	4.6
'p'	0.2675 Not significant		

Mean BMI of ED was 23.2 and ID was 24.4.

The Mean BMI of both groups were not statisticallysignificant (p=0.2675).

Table 4 : ASA

ASA	Group ED		Group ID	
	No	**%**	**No**	**%**
I	17	56.7	19	63.3
II	13	43.3	11	36.7
'p'	0.7921 Not significant			

ASA I among ED group was 56.7% and ID was 63.3%.ASA II among Group ED was 43.3% and ID was 36.7% ASA distribution among two groups were not statistically significant with the 'p' value of 0.7921.

Table 5 :Duration of surgery

Group	Duration of surgery (in minutes)		
	Range	**Mean**	**SD**
Group ED	90-150	112.5	15.9
Group ID	60-160	114.3	20.0
'p'	0.6382 Not significant		

Mean Duration of surgery in ED was 112.5 minutes and in ID was 114.5 minutes . Duration of surgery among two groups were not statistically significant.(p=0.6382).

EFFICACY OF THE TWO ROUTES

Table 6: Sensory onset at T10

Group	Sensory onset at T 10 (in minutes)		
	Range	**Mean**	**SD**
Group ED	6-10	7.23	1.07
Group ID	10-15	12.67	1.79
'p'value	**0.0001** **Significant**		

Mean duration for sensory onset at T10 level in ED group was 7.23±1.07 minutes and in the ID group it was 12.67±1.79 minutes

Table 7: Level of maximum sensory block

Sensory block level	Higher level in			
	Group ED		Group ID	
	No	%	No	%
T2	-	-	-	-
T4	24	80	24	80
T5	3	10	3	10
T6	3	10	3	10
T8	-	-	-	-
T9	-	-	-	-

The highest level of sensory block in themajority number of cases in both groups was at T4 level.

Table 8: Time for 2 segment regression

Group	**Time for 2 segment regression (in minutes)**		
	Range	**Mean**	**SD**
Group ED	60-100	83.7	12.3
Group ID	35-50	43.2	4.3
'p'Value	**0.0001 Significant**		

Mean duration of two segment regression in ED group was 83.7±12.3 minutes and in Group ID it was 43.2±4.3 minutes .This is statistically significant bwith a p value of 0.0001.

Table 9 :Onset of motor block

Group	Onset of motor block (in minutes)		
	Range	**Mean**	**SD**
Group ED	8-14	10.7	1.5
Group ID	8-15	10.2	1.7
'p'Value	0.1218 Not significant		

Mean duration for onset of motor block in ED group was 10.7±1.5 minutes and in ID group was 10.2±1.7 minutes .There is no statistical significance between two groups .(p=0.1218).

Table 10 :Time for complete motor block

Group	Time of complete motor block (in minutes)		
	Range	**Mean**	**SD**
Group ED	11-16	14.4	1.2
Group ID	10-18	14.2	1.8
‘p’value	0.9514 Not significant		

Mean duration for complete motor block was 14.4±1.2minutes in ED group and 14.2±1.8 minutes in ID group .There is no significantstatistical difference between the two groups.

Table 11 : Time for first rescue analgesia

Group	**Time for first rescue analgesia (in minutes)**		
	Range	**Mean**	**SD**
Group ED	135 - 170	158.1	13.4
Group ID	60 - 105	69.7	7
'p'value	**0.0001 Significant**		

Mean duration of time for rescue analgesia in ED group was 158.1±13.4 minutes and in ID group itwas 69.7±7 minutes.The duration for the first rescue analgesic was longer in the ED group when compared to the ID group which is statistically significant with a p value of 0.0001.

CHANGES IN VITAL PARAMETERS

Table 12 : Changes in systolic blood pressure

SBP at	SBP mmHg				'p'	Significance
	Group ED		Group ID			
	Mean	SD	Mean	SD		
2 minutes	116.6	9.2	116.9	9.2	0.8787	Not significant
5 minutes	101.9	6.3	93.8	4.6	0.0001	**Significant**
10 minutes	93.6	4.3	91.7	3.4	0.044	**Significant**
15 minutes	91.9	3.1	101.9	6.3	0.0001	**Significant**
30 minutes	102.2	4.7	105.1	5.4	0.0477	**Significant**
45 minutes	111.9	8.1	111.9	8.1	1.0	Not significant
60 minutes	115.1	8.9	115.1	8.9	1.0	Not significant
90 minutes	116.7	8.0	117.9	8.9	0.7328	Not significant
120 minutes	125.6	8.1	124.9	7.4	0.7385	Not significant

The mean systolic blood pressure at 5 and 10 minutes (101.9 ± 6.3 and 93.6± 4.3)were lower in the ID group when compared to the ED group (93.8± 4.6 and 91.7± 3..4) This was statistically significant.But at 15 and 30 minutes, the mean SBP was lower in the ED group (91.6 ±3.1 and102.2±4.7) when compared to the ID group (101.9± 6.3 and 105.1 ±5.4) The values are statistically significant.At all other times, the SBP was comparable in both the groups.

Table 13 : Changes in Diastolic blood pressure

DBP at	DBP of				'p'	Significance
	Group ED		Group ID			
	Mean	SD	Mean	SD		
2 minutes	81.1	7.7	81.1	7.7	1.0	Not significant
5 minutes	70.6	7.4	67.7	7.5	0.1928	Not significant
10 minutes	65.5	7.1	64.3	6.0	0.6448	Not significant
15 minutes	65.5	6.0	71.5	7.3	0.0009	**Significant**
30 minutes	68.3	6.0	68.1	6.6	0.7481	Not significant
45 minutes	72.4	4.9	70.9	5.1	0.2507	Not significant
60 minutes	72.8	5.2	71.7	4.8	0.4328	Not significant
90 minutes	71.5	5.3	73.5	5.2	0.1405	Not significant
120 minutes	74.0	5.0	75.7	5.2	0.1721	Not significant

Mean value for Diastolic Blood pressure in ED grou at 15 minute 65.5 ±6 was significantly lower than ID group at 15 min (71.5±7.3).

Table 14 : Changes in pulse rate

Pulse Rate at	Pulse Rate of				'p'	Significance
	Group ED		Group ID			
	Mean	SD	Mean	SD		
2 minutes	97.7	13.5	99.0	13.0	0.7389	Not significant
5 minutes	69.4	5.9	66.4	5.7	0.0596	Not significant
10 minutes	69.2	3.6	66.5	3.0	0.0019	**Significant**
15 minutes	67.3	4.2	66.6	4.6	0.9349	Not significant
30 minutes	69.8	6.1	68.6	4.8	0.5049	Not significant
45 minutes	73.7	5.2	71.7	4.8	0.1108	Not significant
60 minutes	75.6	4.9	75.1	4.1	0.7665	Not significant
90 minutes	86.3	4.0	84.8	4.3	0.1124	Not significant
120 minutes	85.9	4.8	84.7	4.3	0.3214	Not significant

The decrease in pulse rate was comparable in both groups, except at 10minutes the decrease in pulse rate in ID group was more which is statistically significant.

Table 15 :Changes in Respiratory Rate

Respiratory Rate at	RespiratoryRate of				'p'	Significance
	Group ED		Group ID			
	Mean	SD	Mean	SD		
2 minutes	15.6	0.8	14.1	0.3	0.0001	**Significant**
5 minutes	14.3	0.65	13.7	0.79	0.0038	**Significant**
10 minutes	14.17	0.53	13.8	0.61	0.0184	**Significant**
15 minutes	14.17	0.46	14.67	0.76	0.0025	**Significant**
30 minutes	14.07	0.25	14.33	0.92	0.0318	**Significant**
45 minutes	14.03	0.18	13.97	0.41	0.5703	Not significant
60 minutes	14.13	0.43	14.47	2.01	0.0007	**Significant**
90 minutes	14.73	0.78	14.67	1.15	0.2756	Not significant
120 minutes	15.27	1.01	15.27	1.28	0.9235	Not significant

Respiratoryrate in both ED and ID groups were between 14 and 16/min. There was no decrease in respiratory rate noticed through out the procedure, but there were statistically significant differences between the two groups.

Table 16: Changes in SPO_2

SPO_2 at	SPO_2 of				'p'	Significance
	Group ED		Group ID			
	Mean	SD	Mean	SD		
2 minutes	99.0	0.53	99.0	0.37	1.0	Not significant
5 minutes	99.0	0.37	99.3	0.6	0.0781	Not significant
10 minutes	99.0	0.37	99.2	0.71	0.1355	Not significant
15 minutes	98.97	0.41	99.17	0.65	0.1397	Not significant
30 minutes	99.0	0.45	98.97	0.41	0.767	Not significant
45 minutes	98.97	0.41	98.8	0.55	0.1711	Not significant
60 minutes	98.97	0.49	99.13	0.57	0.2209	Not significant
90 minutes	99.0	0.37	99.0	0.45	1.0	Not significant
120 minutes	98.93	0.45	99.2	0.63	0.0945	Not significant

Mean value for SPO_2 throughout the procedure was between 98 and 99.There was no significantstatistical difference between ED and ID groups

Table 17 : Sedation Score

Sedation Score at	Sedation Score of				'p'	Significance
	Group ED		Group ID			
	Mean	SD	Mean	SD		
2 minutes	1.13	0.35	1.33	0.48	0.0694	Not significant
5 minutes	1.37	0.49	1.8	0.66	0.0093	**Significant**
10 minutes	2.77	0.68	3.1	0.61	0.0477	**Significant**
15 minutes	2.7	0.68	3.1	0.65	0.0082	**Significant**
20 minutes	2.6	0.5	2.97	0.61	0.0202	**Significant**
25 minutes	2.4	0.5	2.53	0.63	0.3372	Not significant
30 minutes	2.3	0.54	2.43	0.5	0.3539	Not significant
45 minutes	2.1	0.48	0.233	0.48	0.0736	Not significant
60 minutes	1.93	0.52	2.17	0.53	0.0908	Not significant
90 minutes	1.63	0.56	1.8	0.55	0.2458	Not significant
120 minutes	1.5	0.57	1.53	0.57	0.8071	Not significant

The sedation score at 5, 10, 15 and 20 minutes were higher in the ID group and it was statistically significant. At all other times, the sedation score were comparable in both the groups.

Table 18 : Side effects

SIDE EFFECTS	NO OF CASES		'p' VALUE
	GROUP ED	GROUP ID	
Dizziness	1	0	0.987
Dry mouth	3	6	0.876
Shivering	1	0	0.562
Respiratory depression	0	0	0
Nausea	2	3	0.591
Hypotension	4	5	0.560
Bradycardia	3	6	0.876

The incidence of side effects like dizziness, shivering ,head ache , dry mouth, nausea, hypotension and bradycardia were comparable in both groups and were statistically insignificant.

REVIEW OF LITERATURE

1) Dexmedetomidine for the prevention of shivering during spinal anaesthesia. Clinics. 2011 July; 66(7): 1187–1191. BurhanettinUsta, MuhammetGozdemir,Demrcioglu,BunyaminMuslu,HuseyinSertand Andan Yaldiz. This study is conducted to evaluate the efficacy of dexmedetomidinein the control of shivering during spinal anaesthesia with hyperbaric bupivacaine for elective minor surgeries. The results were the intensity of shivering was lower in dexmedetomidine group than saline group. The time from baseline to onset of shivering 10 min in group dexmedetomidine whereas 15 min in group saline. It has been concluded that Intravenous dexmedetomidine infusion is effective in the prevention of shivering and moderate sedation in patients undergoing spinal anaesthesia.

2) A comparative study of duration of postoperative analgesia between epidural bupivacaine and epidural clonidine plus bupivacaine in lumbar laminectomy surgery under general anaesthesia

Journal of Indian medical association 2011 April;109(4):230-3.

Niyogi S, Santra S, Chakraborty J, Chakraboty S, Mandal S. Department of Anaesthesiology,BIN,Kolkata.They conducted a Randomised

prospective double blind placebo controlled study for patient undergoing laminectomy under general anaesthesia.

They have concluded that no clinically significant difference was found between two groups in pulse rate, Mean arterial pressure, respiratory rate, Spo2, and motor block but adding small amount of alpha 2 agonist as an adjuvant in epidural route with bupivacaine providing better sedation and prolong post operative analgesia and improved patent satisfaction without any side effects.

3) Synergistic effect between dexmedetomidine and 0.75% Ropivacaine in epidural anaesthesia. Rev Assoc Med Bras 2008 Mar-April 54(2):110-5. Salgado PF, SabbagAT, Silva PC BrienzeSL, DaltoHP, Braz JR, Modolo NS.This study aimed to evaluate clinical characteristics of epiduralanesthesia performed withropivacaine added with dexmedetomidine. The results of the study showsthatepidural dexmedetomidine had no effect on onset time and upper level for sensory analgesia but it prolonged sensory block duration significantly Motor block intensity and duration increased and also post operativeanalgesia. Values of bispectral index were lower in epidural dexmedetomidine group. There was no difference in incidence of hypotension and bradycardia. Incidence of side effects including shivering, vomiting, and decreased in spo2 values less than 90% were low.

4) Comparative evaluation of Dexmedetomidineand fentanyl for epidural analgesia in lower limb surgeries Saudi Journal of anaesthesia 2011 october ;5(4): page 365- 370.

BajawaSJ ,Arora V, Kaur J, Singh A, Parma S S. This study aimed at to compare the hemodynamic, sedative, and analgesia potentiating effects of epidural fentanyl and dexmedetomidine with ropivacaine. . The results shows that the onset of sensory analgesia at T10 (7.12±2.44vs 9.14±2.94) and the time for complete motor blockade (18.16±4.52 vs22.98±4.78) was significantly earlier in the Ropivacainedexmedetomidine group. Post operative analgesia was prolonged significantly in the ropivacaine and dexmedetomidine group. (366.62±24.42) and low consumption of local anaesthetics (76.82±14.28vs 104.35±18.96) during epidural top ups post operatively. Sedation scores were better in ropivacaine and dexmedetomidine group. It was statistically significant on comparison. Occurance of nausea and vomiting was significantly high in ropivacaine and fentanyl group but drynessof mouth is significantly high in RD group. ($p<0.05$).It has been concluded that dexmedetomidine seems to be a better alternative to fentanyl as an epidural adjuvant.It provides better stable hemodynamics, early onset of sensory block, prolonged post op analgesia, lower requirement of post operative local anaesthetics for epidural analgesia and better sedation score.

5) Comparison of the activity and reliability of intravenous administration of midazolam and dexmedetomidine on sedation level under Epidural anaesthesia. Agri 2010 July;22(3) page-121-30.

Kuzucuoglu T, Bolukbasioglu I, Arslan G, Yuce E, Ayaz B , Dr.LutfiKidar.They conducted a study to assess the sedative effects of midazolam and dexmedetomidine and hemodynamic stability under epidural anaesthesia.Result shows that in dexmedetomidine group, mean arterial pressure was significantly higher and pulse rate was significantly lower than in midazolam group. It has been concluded that both drugs provided good sedation without respiratory depression, stable hemodynamics and alertness with good patient cooperation.

6) Effect of Dexmedetomidine on duration of anaesthesia and wakefulness in Bupivacaine epidural Block.

Europian Journal of Anaesthesiology 2007 Jun;24(6):535-40.

E pub 2007 Jan 23.Coskuner I, TekinM, KatiI, YagumurC, Elcicek K. Yagumur C. They conducted a study to assess the effects of Intravenous dexmedetomidine in prolonging the duration of epidural anaesthesia with bupivacaine and level of sedation and sideeffects compare to saline group.

60 patients in ASA I and II were included in the study.They were divded into two groups.The results of the study shows that the duration of sensory analgesia was significantly longer, decreased heart rate was significantly lower in the dexmedetomidine group. The bispectral index values were lower in the dexmedetomidine group than in the saline.

Conclusion is intravenous route of dexmedetomidine prolonged the duration of epidural anaesthesia, provided sedation with few sideeffects.

7) Dexmedetomidine and clonidine in epidural anaesthesia

A comparative evaluation. BaiwaSJ, Baiwa SK, Kaur J, AroraV, Gupta S, Kulshrestha A, Singh A, Parmar S Singh G ,Gorava S.

Indian Journal of Anaesthesiology March 55(2) page 116-121.

This study conducted to compare the clinical profile and efficacy of dexmedetomidine and clonidine in epidural anaesthesia.It has been concluded that sedation score with dexmedetomidine group is significantly higher than clonidine group ($P< 0.05$). Regarding the side effectsof both groups shows that there were incidence of nausea and dry mouth was noticed but not statistically significant. Dexmedetomidine is a better epiduraladjuvant compared to clonidine for providing early onset of sensory block, better sedation during intra operative period and prolong post operative analgesia.

8) Epidural anaesthesia with Intravenous dexmedetomidine sedation in the successful anaesthetic management of MRI- guided focused ultrasound ablation of early prostatic cancer. NgAT, JP Yam PC. Journal of Anaesthesiology 2011 october 25(5) page 756 -759

In this report patients posted for MRI guided focused ultrasound ablation of prostatic cancer taken under epidural anaesthesia with intravenous dexmedetomidine. It provides better sedation score without causing respiratory depression or hemodynamic instability and also useful in preventing shivering..

9) Dexmedetomidine as an adjuvant to ropivacaine prolongs peripheral nerve Block. D Marhofer , S.C. Kettner , S.Pills, M.Weber and M Zeilliger. They conducted this study in volunteers to assess efficacy of adding Dexmedetomidine with ropivacaine in peripheral nerve block versus systemic administration of dexmedetomidine in peripheral nerve block by Ropivacaine .It has been concluded that profound prolongation of nerve block was noticed if dexmedetomidine added with ropivacainethan systemic administration of dexmedetomidinebut no difference notified in onset of sensory block but motor block onset is faster in ropivacaine with dexmedetomidine group .

10) Epidural Dexmedetomidine in regional anaesthesia to reduce anxiety. SA oriole –Lopez, A Maldono- sanchez , CE Hernandez- Bernal, JA Castelazo-Arredono , L Moctezuma . Revista Mexicana de Anestesiologia vol.31 No.4 october-Dcember2008 :page 271-277.

The use of dexmedetomidine as adjuvant with lignocaine given in epidural for regional anaesthesia with adrenaline give the sedation score of 3 at 5 min, 3-4 at 15 to 90 min in majority of population. All patient were hemodynamic stablewithout respiratory depression.

11) Intravenous Dexmedetomidine prolongs Bupivacaine spinal anaesthesia not midazolam.

Canadian Journal of anaesthesiology (2010) 57 page-39-45.

In this randomized double blind clinical study and a comparison was made with midazolam to assess the effect of intravenous dexmedetomidine as premedication on spinal anaesthesia duration as well as on sedation and post operative analgesia.Results shows that in Dexmedetomidine group 7 patients out of 25 required analgesia in first post operative day significantly less than placebo group 16 patients required analgesia and in midazolam group 15 patients require analgesia ($p<0.05$).

12) Intravenous dexmedetomidine prolongs Bupivacaine spinal anaesthesia. M.E.J.Anaesthesia 2009:20(2).

Supplementation of spinal anaesthesia with intravenous dexmedetomidine produces significantly longer sensory and motor block than spinal analgesia alone. All patient reached better sedation levels that enabled patients cooperation and better operating conditions for the surgeon without respiratory depression.

13) Effect of epidural demedetomdine on intraoperative awareness and post operative pain after one lung ventilation. Acta anaesthesiologyScand 2010 Jul 54 (6) page 703-709. Elhakin M, Abdelfattach H

Abdelhamid D,ElshafeiM ,Elsayed A. During combined regional and General anaesthesia they compared the effect of a dexmedetomidine bupivacaine mixture with bupivacaine for thoracic epidural anaesthesia. Results showed that there was a limited decrease in PaO_2 at one lung ventilation in dexmedetomidine group compare with plain bupivacaine group.They concluded that the use of epidural dexmedetomidine as an adjuvant in epidural in general anaesthesia the requirement for anaesthetic drug dose reduced significantly, also produce unawareness state during surgery and prolong post operative analgesia thereby improve oxygenation.

DISCUSSION

The introduction of dexmedetomidine has fascinated anaesthetist due to its wide uses in the field of anaesthesia. Its route of administration and its qualities like analgesic, sedative without causing respiratory depression. Using dexmedetomidine as an alternative to opioid in epidural routeto avoid respiratory depression , pruritis and urinary retention. Clonidine has been used in past in regional anaesthesia as an adjuvant to local anaesthesia due to its faster onset of action , duration of analgesia,dose sparing effect and its hemodynamic stability and its efficacy is comparable with dexmedetomidine which is a more selective alpha2 agonist. Following administration of drug through epidural route it effectively inhibits the transmission of nociceptive impulse at spinal cord level from peripheral surgical stimulation.

It causes sedation,analgesia and improve the quality of epidural anaesthesia. Intravenous administration of dexmedetomidine has its supraspinal effect due to its inhibitory action over locusceruleus.The nor adrenergic innervation of the spinal cord arise from the noradrenergic nuclei present in the brain stem activity.so, the disinhibition of the noradrenergic nuclei lead to descending inhibitory effect on spinal cord thereby prolongs the sensory block duration rather than motor block.

In this study the efficacy of dexmedetomidine is compared between two routes of administrations; the epidural route and the intravenous route.The mean age ,sex distribution,the BMI, the ASA classification and the duration of surgery were comparable in both groups and there was no statistical difference between two groups.

The sensory onset time was shorter in the ED group and the highest level of sensory block in both group was T4 level and the lowest level in ED group was at T6 and in ID group it was at T9. Compare to Intravenous route (12.67±1.79) epidural route(7.23±1.07) has significant earlier onset of sensory block with the 'p' value of 0.0001.Results for onset and duration of sensory analgesia correlated with the study conducted by Bajawa S J, Arora V in Saudi journal Anaesthesia 2011.Regarding Motor block is assessed by Modified bromagescale.The motor block onset and duration are compared between two groups did not have statistical significance with 'p'value of 0.1218. but,this results being differ from the published results in SJA 2011.Probably due to the conduction of sensory fibre may be more inhibited than motor fibre. Mean duration time for two segmental regression in ED group (83.7±12.3)was statistically significant than ID group(43.2± 4.3) with the 'p' value of 0.0001.

Kuzucuoglu in Agri, July 2010 states that intravenous dexmedetomidine as premedication for regional anaesthesia prolong sensory block but the results is not superior to epidural route.

Intraoperativelyalthough thesystolic blood pressure fall occur in both group earlier fall with Intravenous administrated group later fall in blood pressure with epidural route administration.The results were statistically significant. Probably due to decreased sympathetic activity and circulating levels of catecholamines. Regarding Diastolic blood pressure significant difference shown at 15 min withlower diastolic blood pressure in epidural dexmedetomidine.

The decrease in pulse rate in ID group was more than ED group at 10 min which is stasticallysignificant.Bradycardia was treated in 6 patient with Atropine 0.3 mg in patients under ID group comparable with ED group but not significant.

Sedation was assessed with Ramsay sedation score graded from 1 to 6. Most of the patient fell between 2 and 3 grade easily arosable with oral commands. Sedation score significantly higher in Intravenous group between 5 and 20 minutes.

Epidural anaesthesia for lower abdominal surgeries reduces endocrine and metabolic changes due to surgical stress than upper abdominal surgeries. Dexmedetomidine in a concentration producing significant sedation retains the ventilatory response to increasing carbon-di-oxide.A frequently reported side effect of demedetomidine is dry mouth due to decreased saliva production.The changes in ventilation appear to be similar to those observed during natural sleep.The beneficial effect on myocardial oxygen balance has been shown to decrease myocardial ischemia and infarction in cardiac as well as non cardiac surgery. The probability of respiratory depression using anxiolytic is decreased in these patient but level of sedation is achieved with arousable sleep.

SUMMARY

This study was conducted in 60 patients undergoing lower abdominal surgeries under epidural anaesthesia.These patients are divided into two groups with 30 patients in each group between 35 and 65 years assessed under ASA I and II.

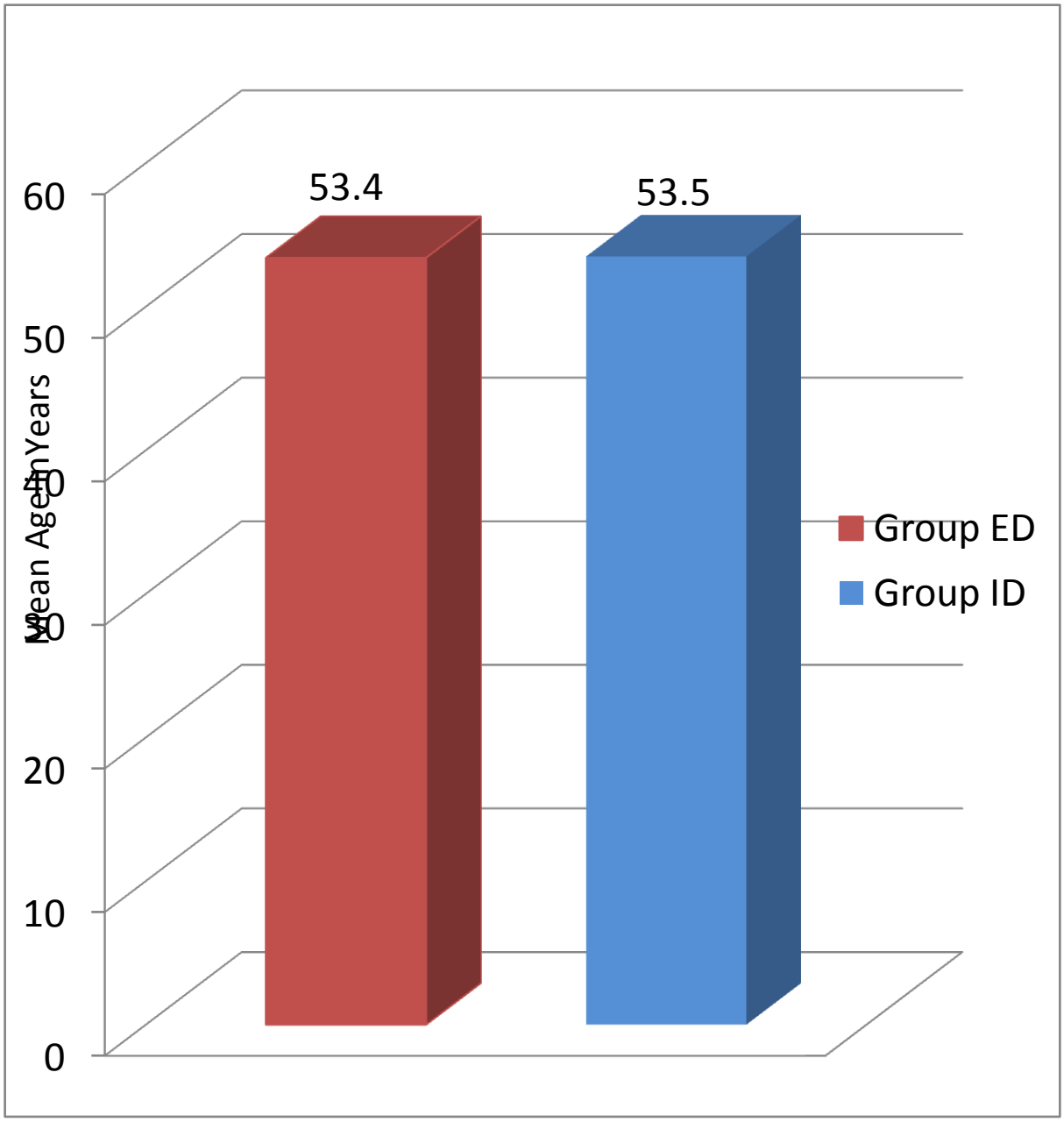
Group ED- Epidural route administration of 2%lignocaine 5mg/kg with Adrenaline 5µg/ml and Dexmedetomidine 1µg/kg

Group ID- Intravenous route administration of Dexmedetomidine 1µg/kg with Epidural route administration of 2% lignocaine 5 mg/kg with Adrenaline 5µg/ml.This study shows epidural route of administration of dexmedetomidine provides earlier onset of sensory block, time for two segment regression was prolonged, time for rescue analgesia is prolonged than intravenous route of administration of dexmedetomidine. Regarding the onset and duration of motor blockade both the groups had similar results. Regarding the sedation observed in the intraoperative period both groups provided sedation but sedation score was higher and statistically significant in the ID group at 5, 10,15,and 20 minutes.The Hemodynamic stability was similar with both groups. The Incidence of complications like nausea,hypotension, bradycardia and drymouth were similar in both groups.

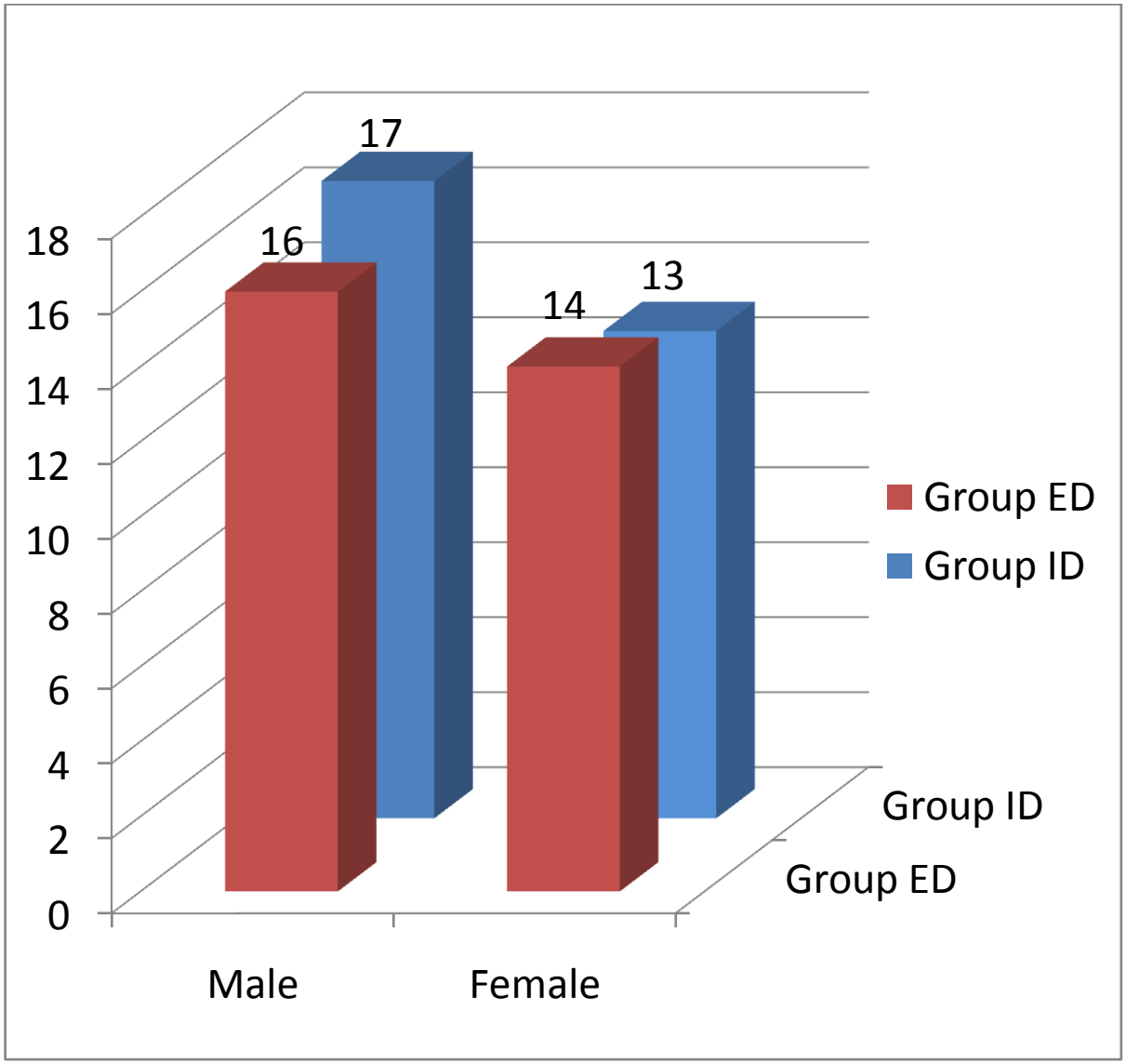
CONCLUSION

The addition of dexmedetomidine as an adjuvant to lignocaine in epidural anaesthesia provides better quality of anaesthesia, and prolongs the duration of action of lignocaine whereas dexmedetomidine when administered intravenously provides better sedation than epidural route.In both of the routes either via epidural or intravenously dexmedetomidine maintains stable cardio-respiratory parameters and patient comfort.

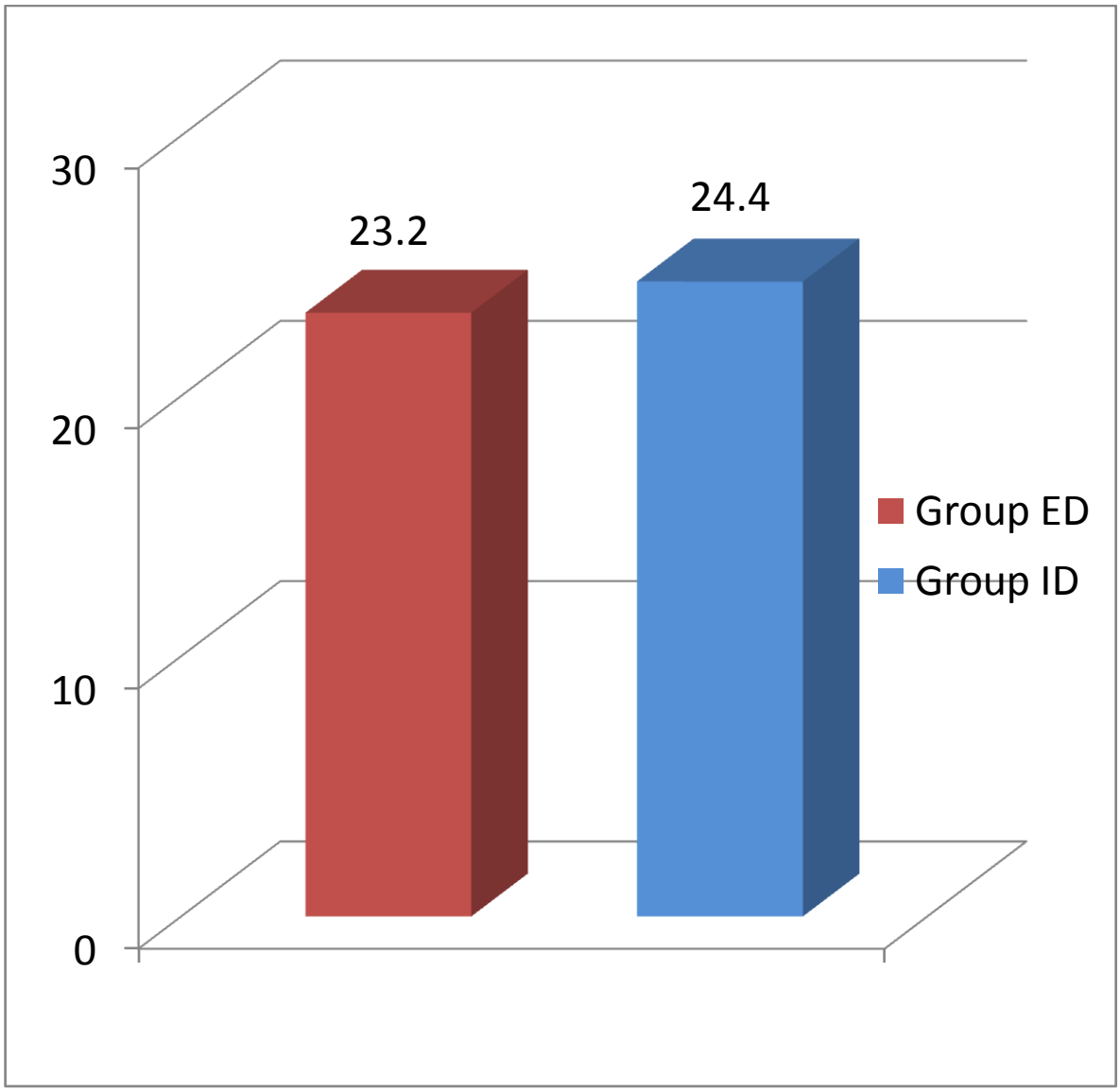
AGE



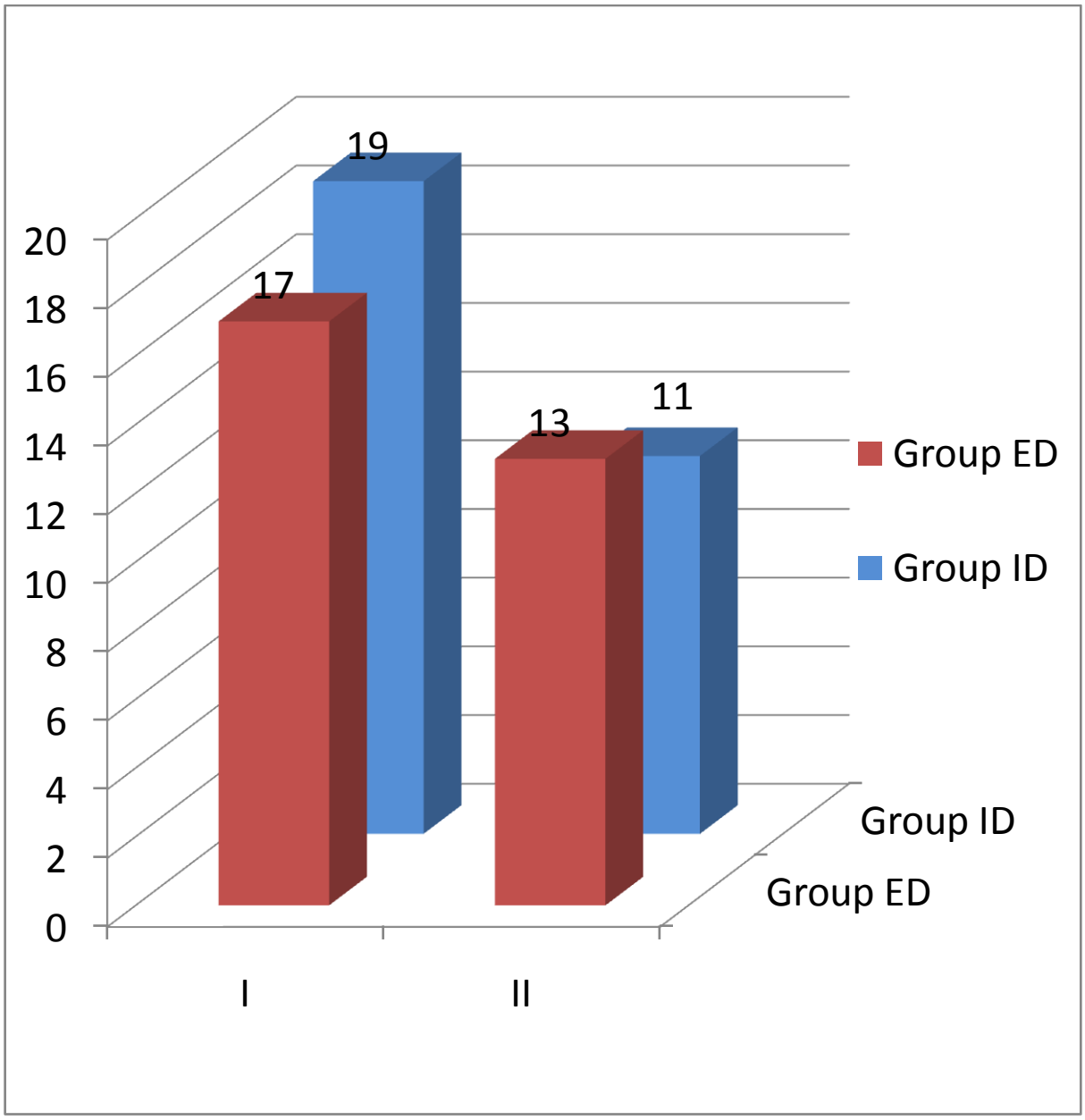
SEX DISTRIBUTION



BMI



A S A GRADE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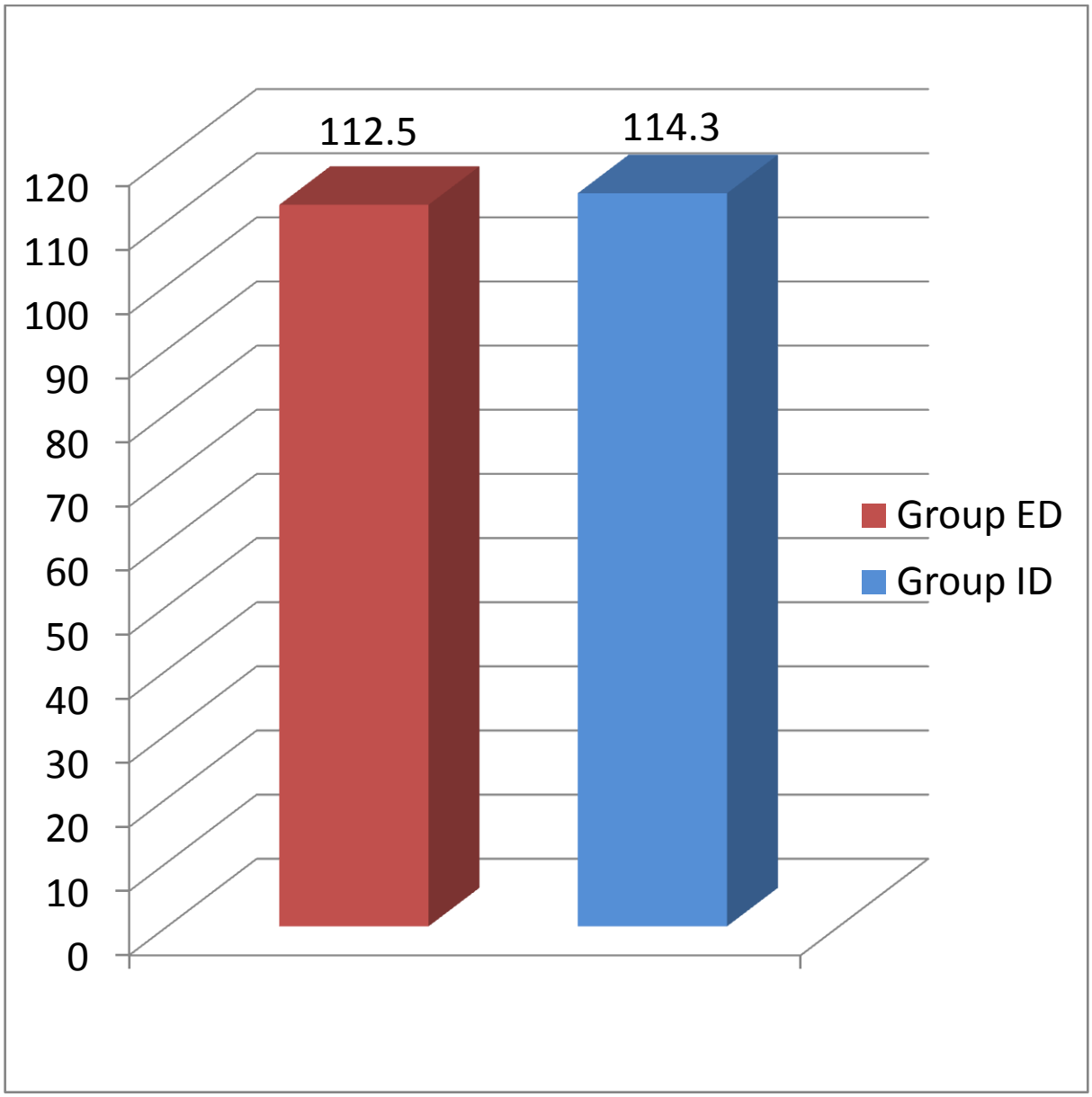
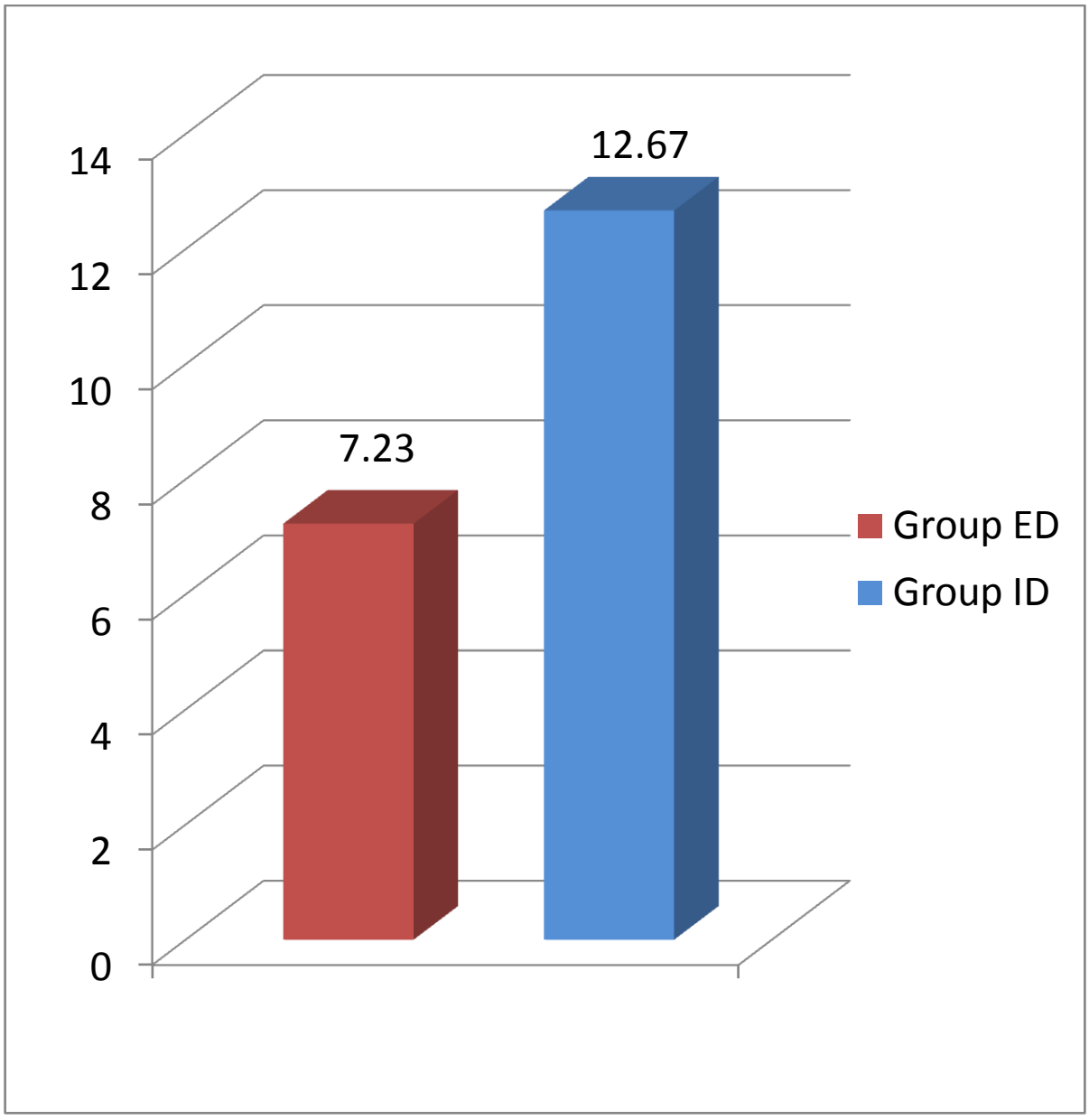
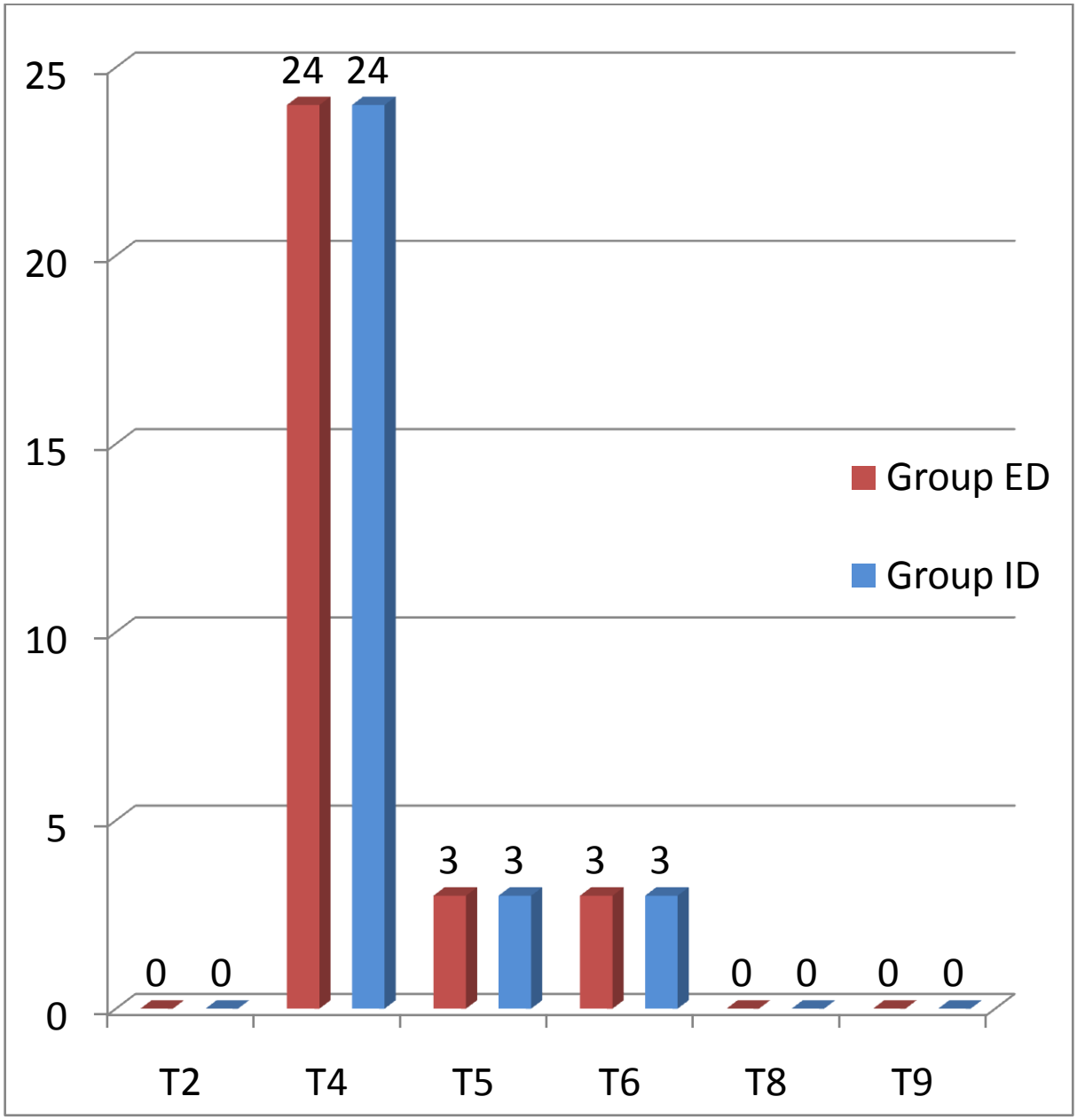
DURATION OF SURGERY



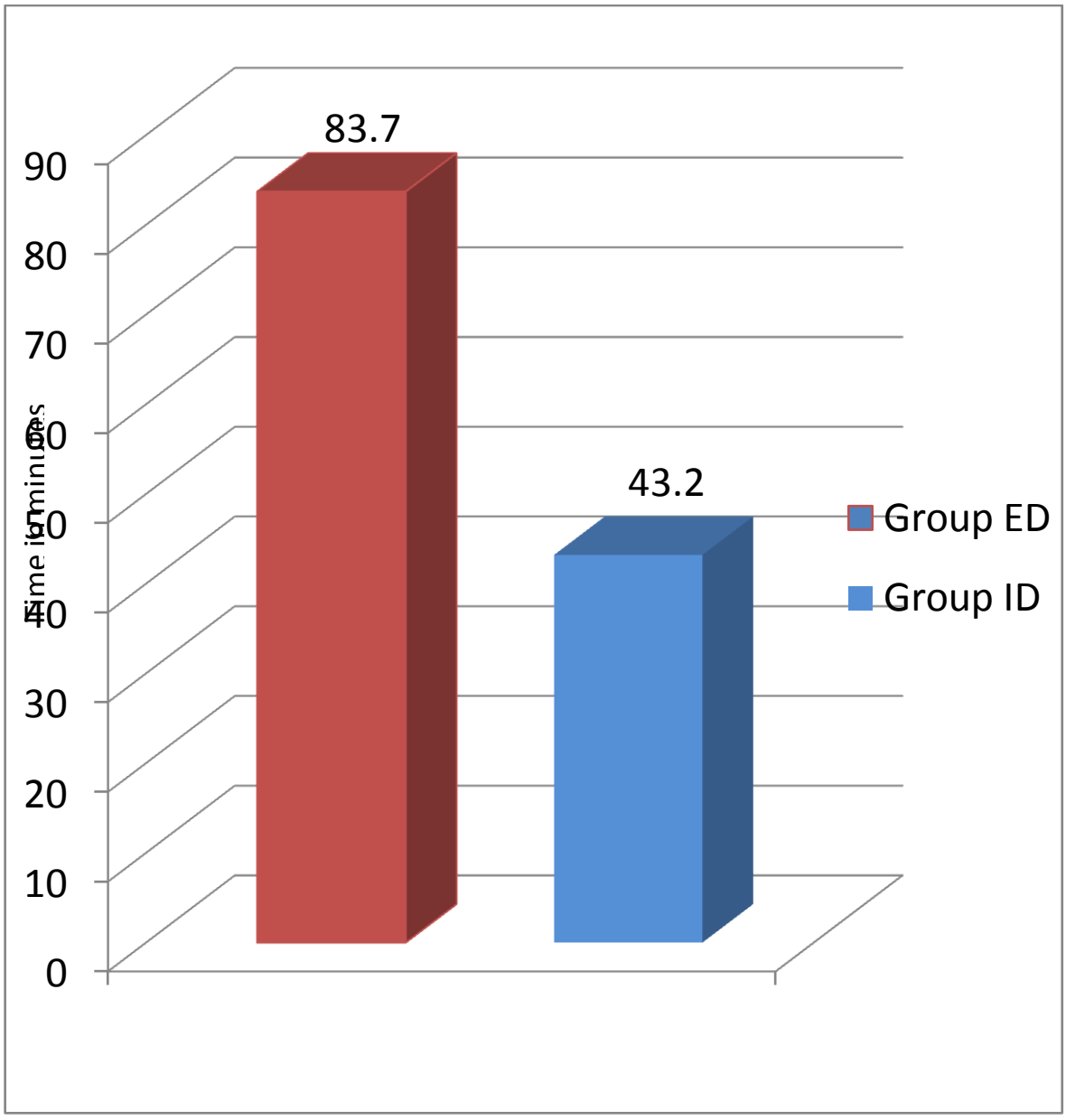
SENSORY ONSET AT T10



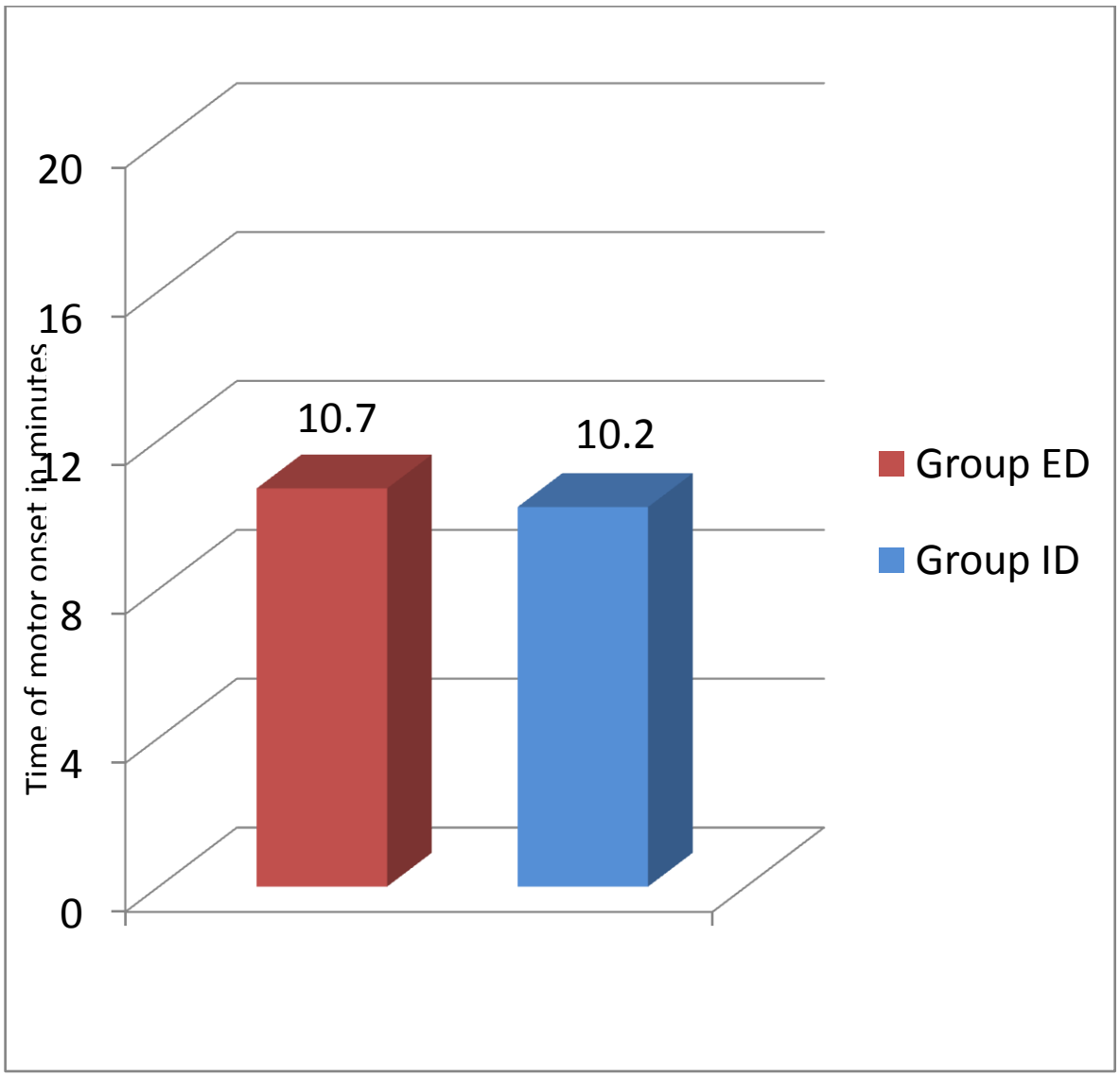
LEVEL OF MAXIMUM SENSORY BLOCK



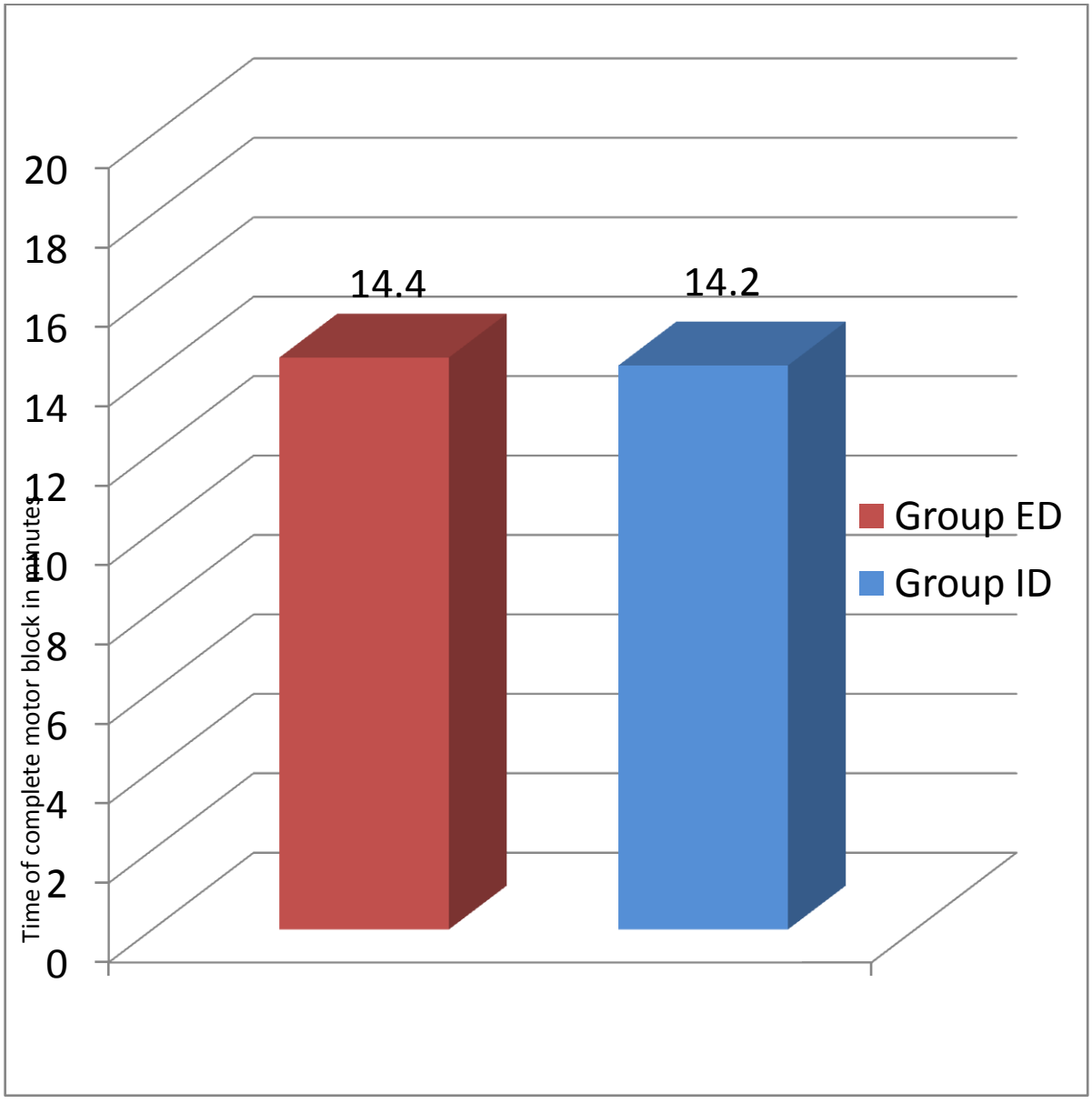
TIME FOR TWO SEGMENT REGRESSION



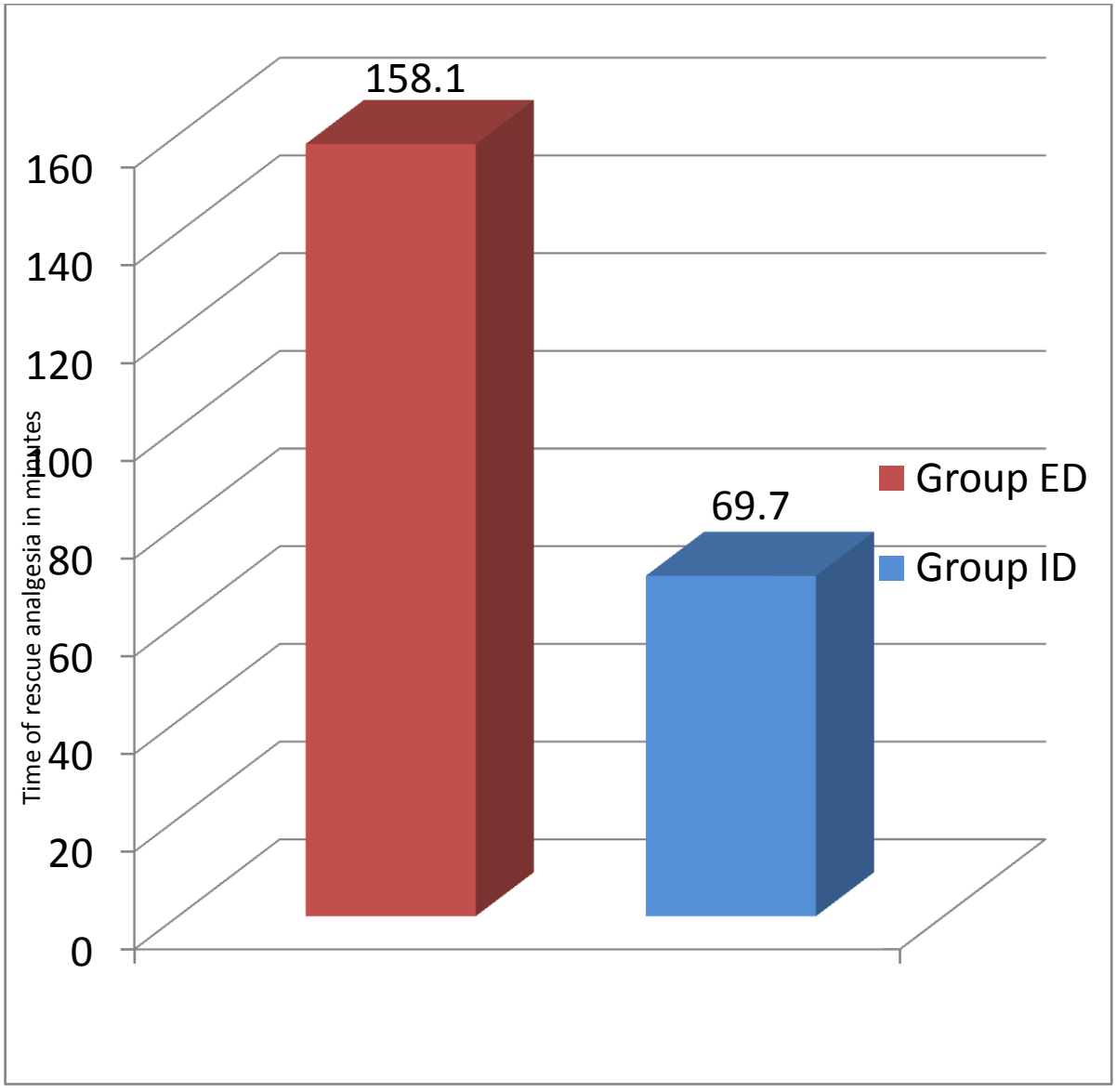
ONSET OF MOTOR BLOCK



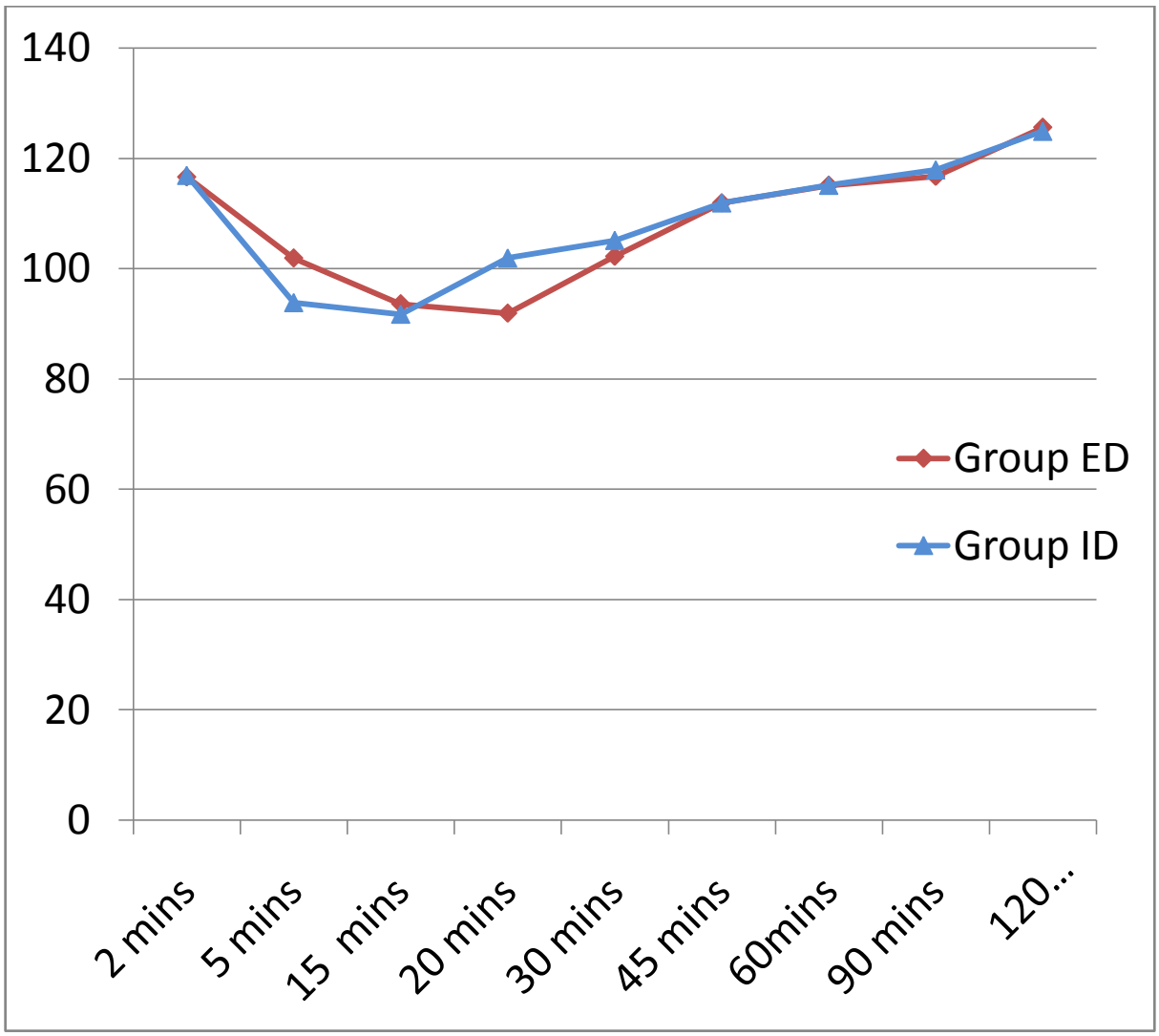
TIME FOR COMPLETE MOTOR BLOCK



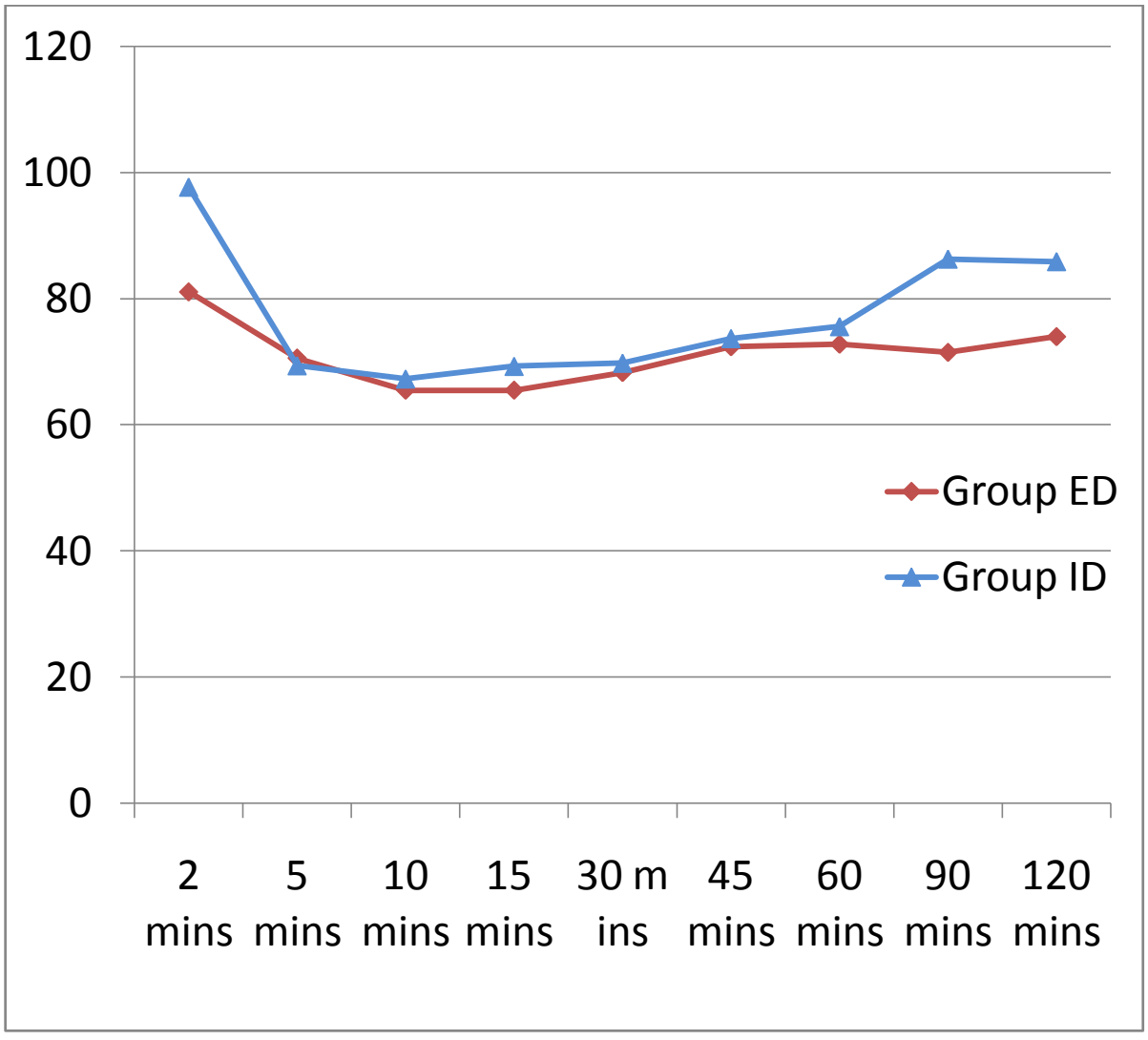
TIME FOR FIRST RESCUE ANALGESIA



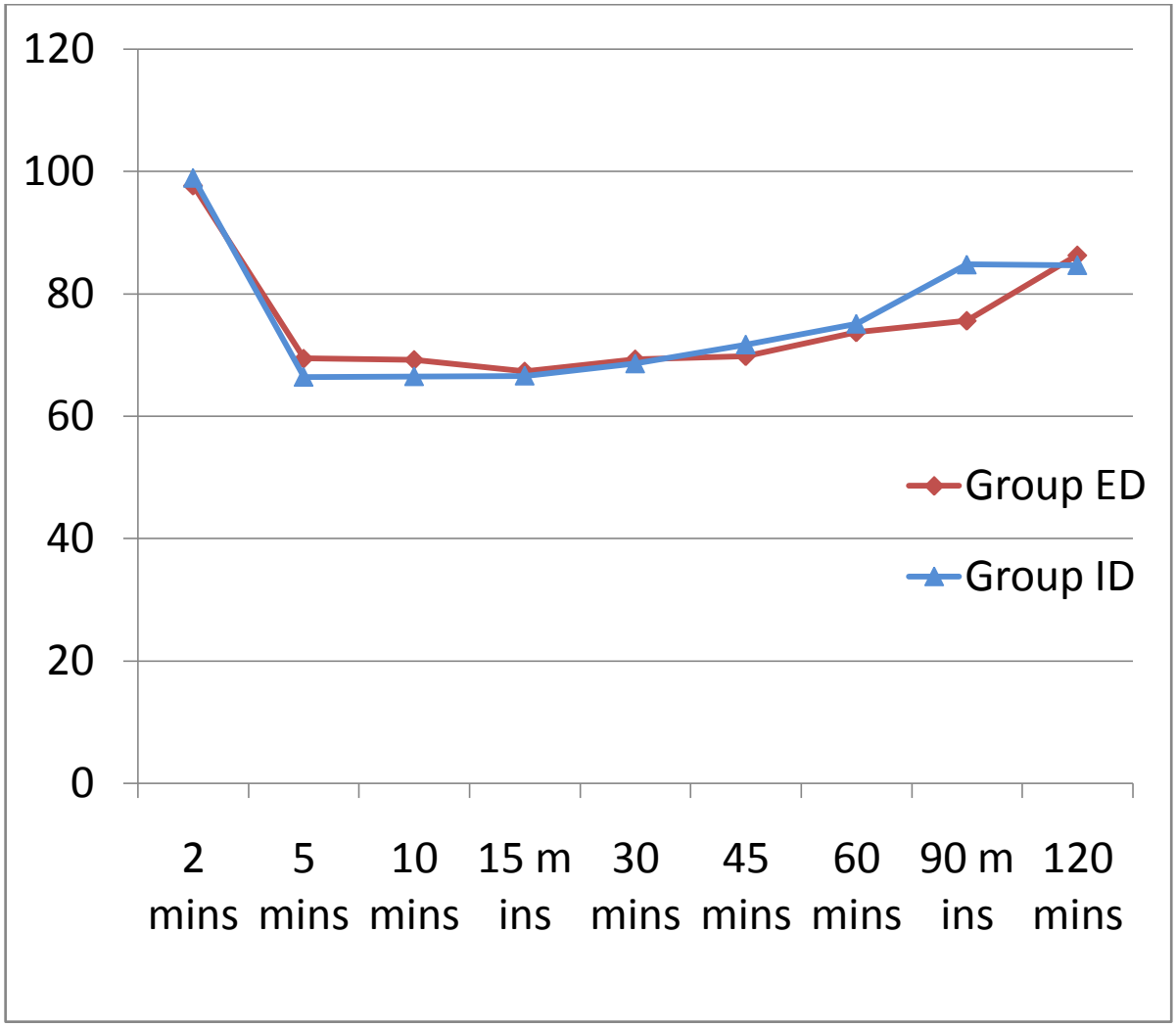
CHANGES IN SYSTOLIC BLOOD PRESSURE



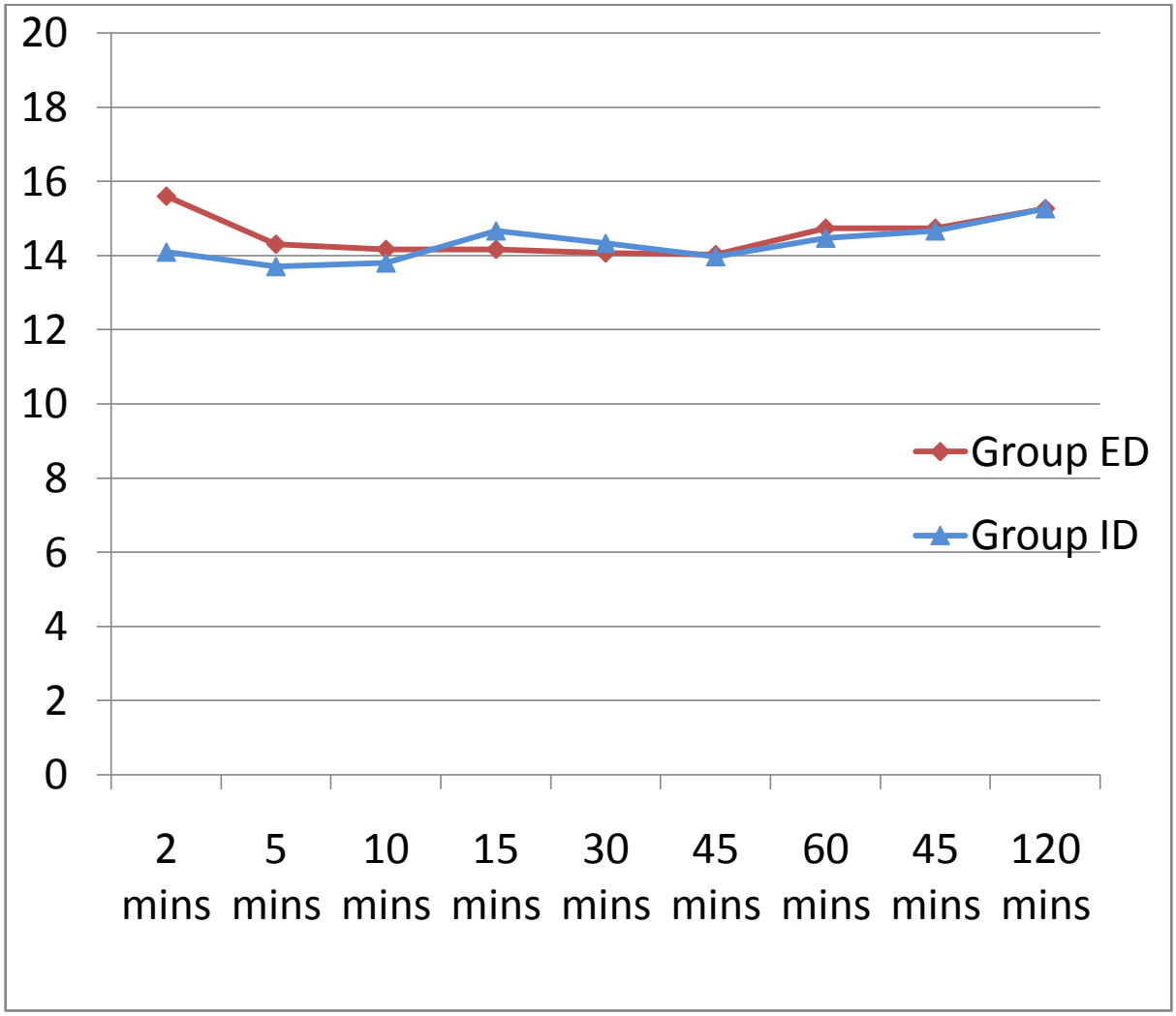
CHANGES IN DIASTOLIC BLOOD PRESSURE



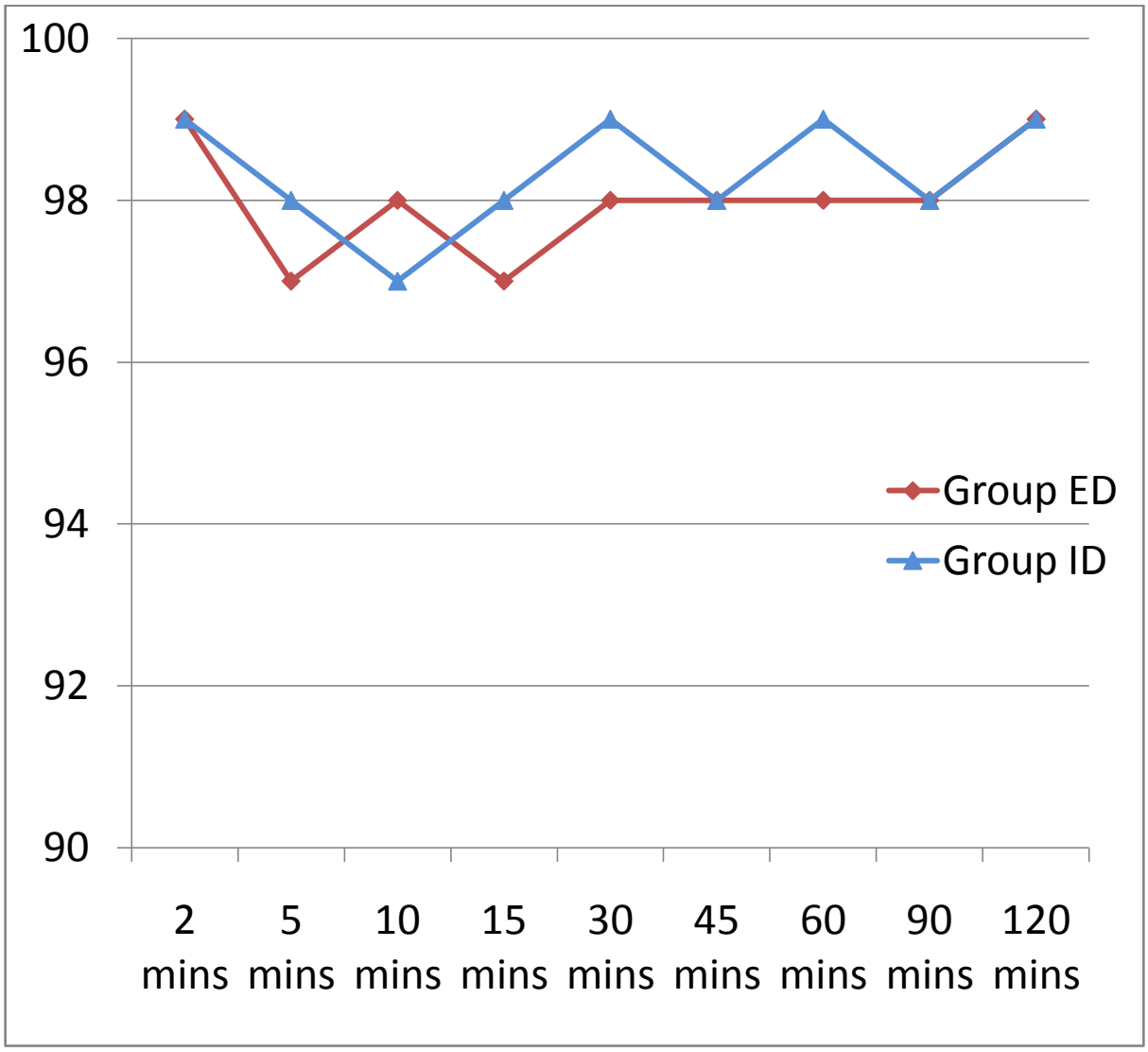
CHANGES IN PULSE RATE



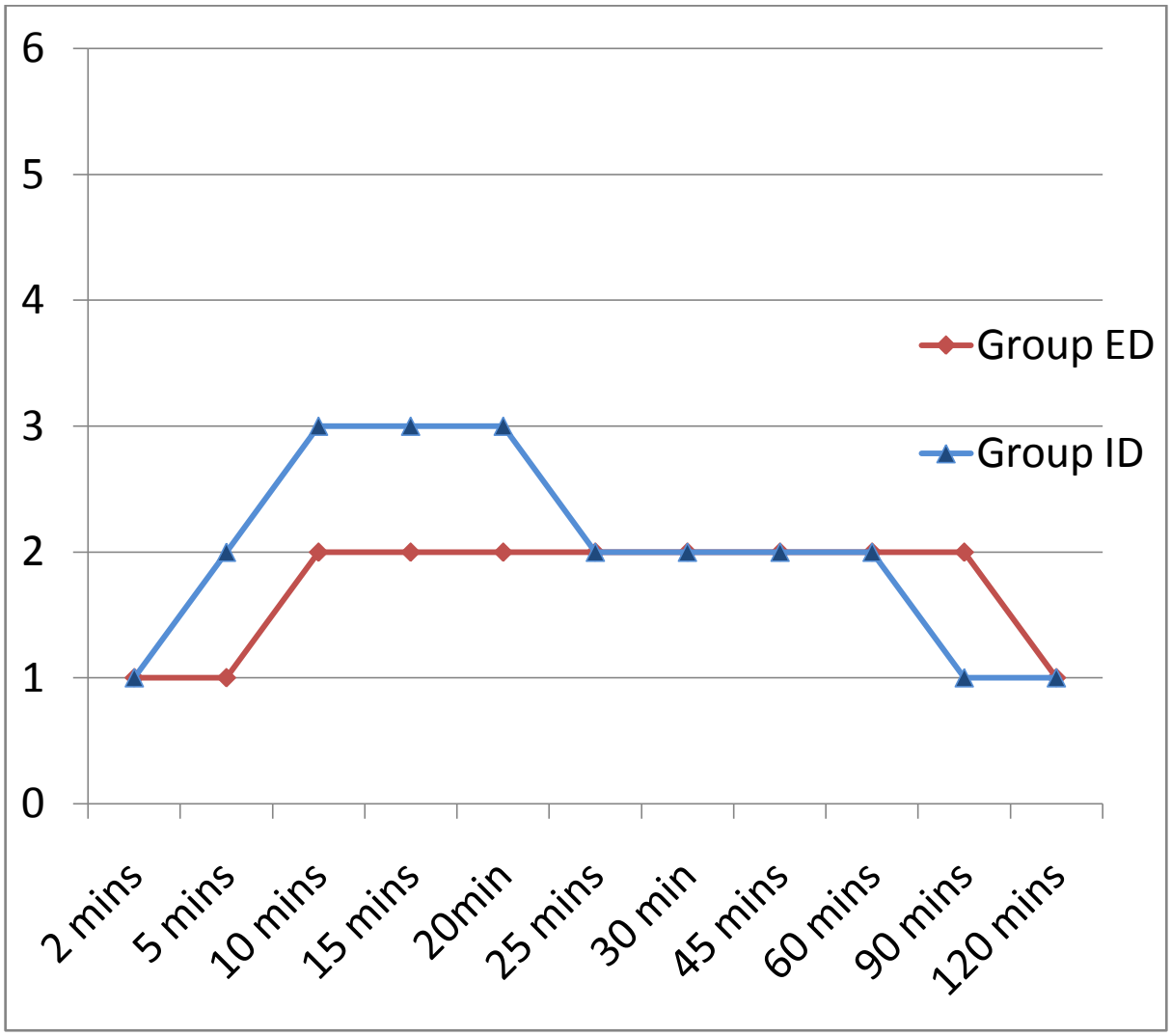
CHANGES IN RESPIRATORY RATE



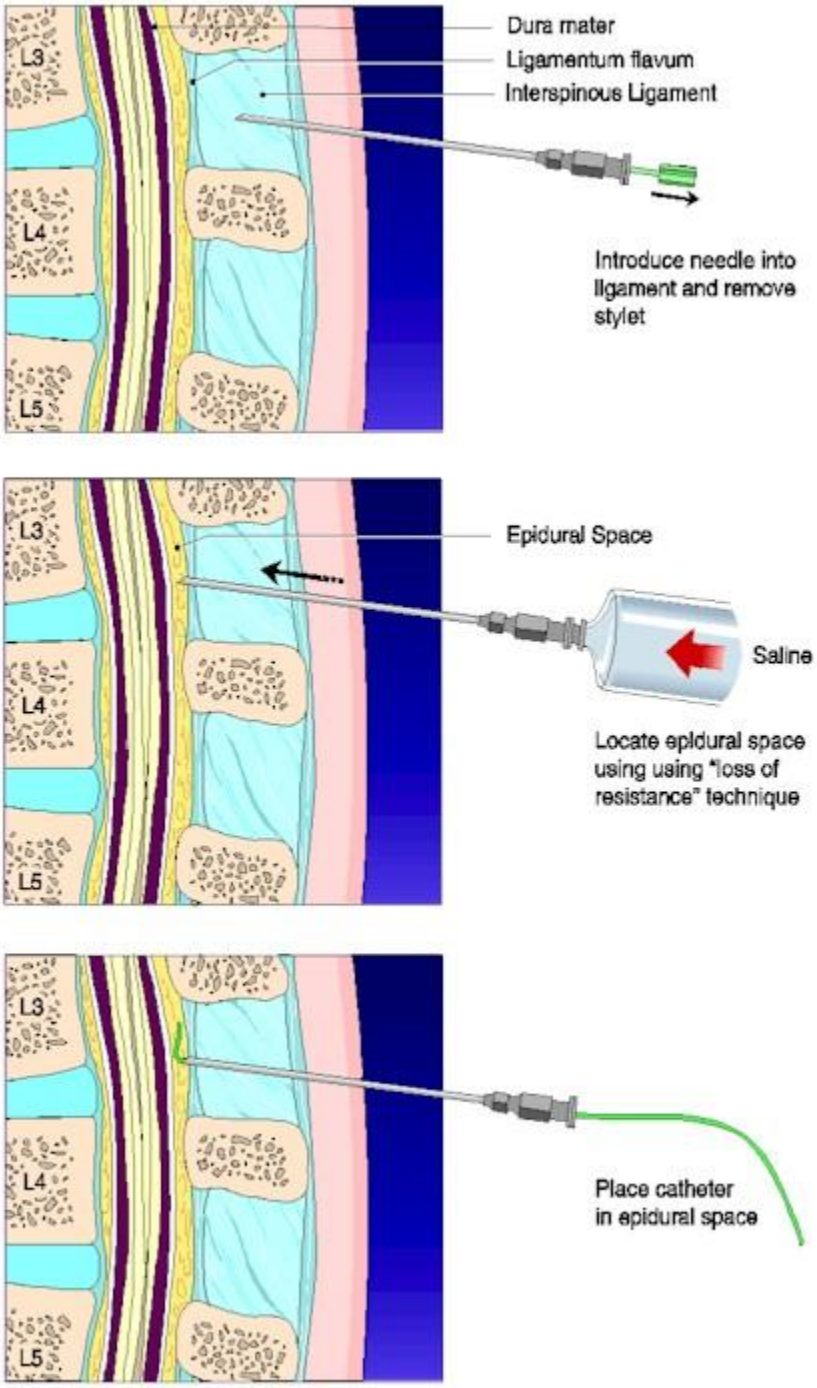
CHANGES IN SPO_2



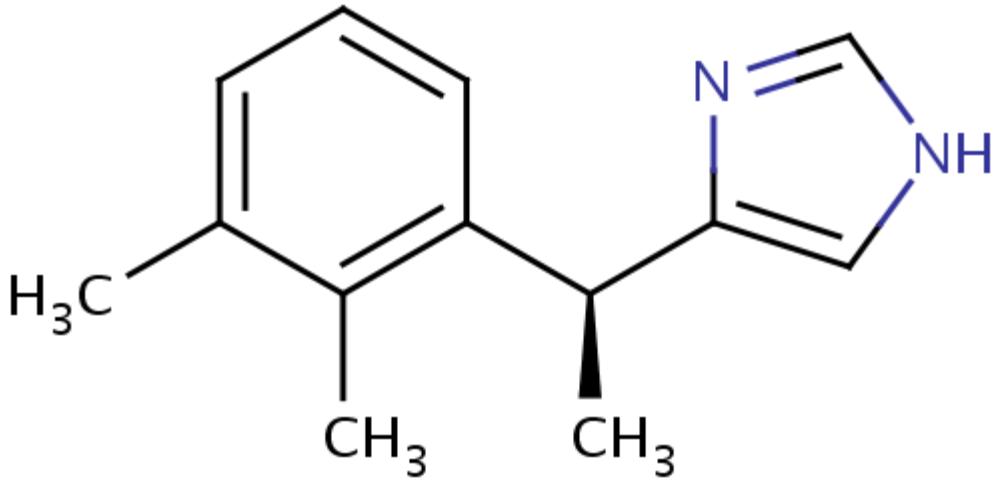
CHANGES IN SEDATION SCORE



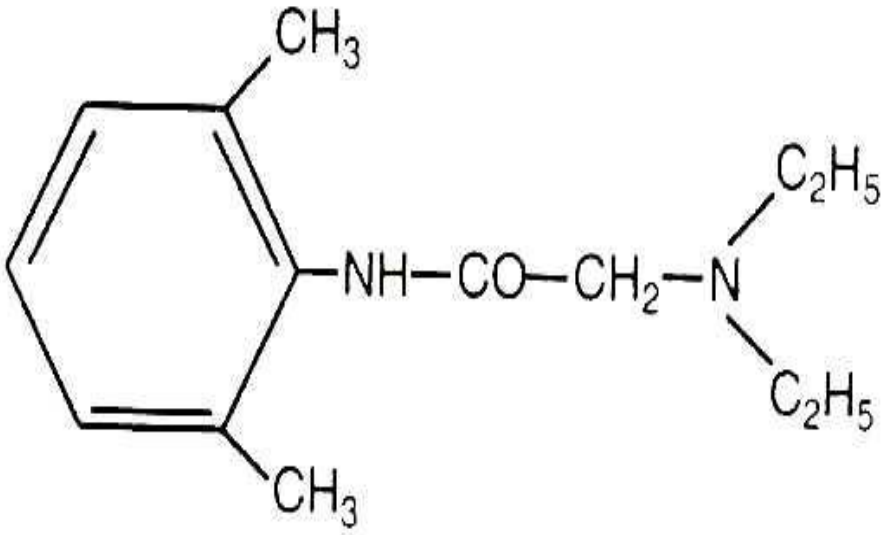
EPIDURAL TECHNIQUE



STRUCTURE OF DEXMEDETOMIDINE



STRUCTURE OF LIGNOCAINE



Ref. No. 5336 /E4/3/2012

Govt. Rajaji Hospital,
Madurai.20. Dated: .08.2012

Institutional Review Board / Independent Ethics Committee.
Dr. N. Mohan, M.S., F.I.C.S., F.A.I.S.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 28.06.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

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1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5.Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6.Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7.Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8.Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9.Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10.Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

21/8/12

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Dr. Sivakumar. S	M.D Anaesth	Epidural vs. intravenous dexmedetomidine.	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance.

She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.

4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

DEAN 13.8.12

To
All the above members and Head of the Departments concerned.
All the Applicants.

DIRECTOR
INSTITUTE OF ANAESTHESIOLOGY
Madurai Medical College &
Govt. Rajaji Hospital
MADURAI 625020.

MASTER CHART GROUP - ED

S. No.	NAME	AGE / SEX	IPNO	BMI	ASA	DIAGNOSIS	PROCUDURE	EPIDURAL SITE	CATHETER TIP	AVERAGE DURATION OF
1	PANDIAN	40/M	76809	20.37	I	B/lIng Hernia	Hernioplasty	L1-L2	T11	90
2	BOOPATHY	45/M	67370	21.66	I	B/lIng Hernia	Hernioplasty	L1-L2	T11	105
3	SHAHUL	60/M	23415	27.08	II	B/L Ing Hernia	Hernioplasty	L1-L2	T11	90
4	MUTHUKUMAR	58/M	87965	27.35	I	B/ling hernia	Hernioplasty	L1-L2	T11	120
5	RAMAN	56/M	23456	25.39	I	B/lIng hernia	Hernioplasty	L1-L2	T11	105
6	RAMU	54/M	56789	23.66	I	B/lIng hernia	Hernioplasty	L1-L2	T11	120
7	KALYANASUNDARI	47/F	96896	25.45	I	paraumblical hernia	Mesh repair	L1-L2	T11	130
8	GANESAN	56/M	26837	19.14	I	B/L Ing hernia	Hernioplasty	L1-L2	T11	120
9	SUSEELA	46/F	38058	16.22	I	B/L Ing hernia	Hernioplasty	L1-L2	T11	110
10	PANDEESHWARI	53/F	27058	18.42	II	incisional hernia	Mesh repair	L1-L2	T11	120
11	PUSHPAM	54/F	86438	16.98	II	incisional hernia	Mesh repair	L1-L2	T11	100
12	THAVASI	63/F	88269	16.78	I	incisional hernia	Mesh repair	L1-L2	T11	105
13	BAKKIYAM	60/F	57054	17.3	II	B/L Ing Hernia	Hernioplasty	L1-L2	T11	120
14	LAKSHMANAN	56/M	96847	18.9	I	B/L Ing Hernia	Hernioplasty	L1-L2	T11	105
15	SATHAPPAN	55/M	48389	21.1	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	90
16	KARUPPUSAMY	55/M	37378	19.77	II	B/ling Hernia	Hernioplasty	L1-L2	T11	90
17	ALAGAMMAL	52/F	84848	16.88	I	incisional hernia	Mesh repair	L1-L2	T11	100
18	MAHALAKSHMI	47/F	95326	18.9	I	umbilical hernia	Mesh repair	L1-L2	T11	105
19	PANDIARAJAN	56/M	86436	19.85	II	B/lIng hernia	Hernioplasty	L1-L2	T11	110
20	KALIYAMMAL	61/F	23568	24.2	I	incisional hernia	Mesh repair	L1-L2	T11	125
21	THANGARASU	41/M	23578	23.18	II	U/L ing.hernia	Hernioplasty	L1-L2	T11	120
22	PANCHAVARNAM	49/F	46778	21.91	II	incisional hernia	Mesh repair	L1-L2	T11	110
23	KALIYAMMAL	51/F	34677	22.5	II	incisional hernia	Mesh repair	L1-L2	T11	120
24	AROKYARAJ	60/M	57890	19.2	II	B/L ing Hernia	Hernioplasty	L1-L2	T11	140
25	KARUPPAIAH	64/M	75435	22.9	I	B/L ing Hernia	Hernioplasty	L1-L2	T11	150
26	SARATHAMBAL	47/F	65467	21.7	II	incisional hernia	Mesh repair	L1-L2	T11	90
27	RAJJAMMAL	48/F	56788	20.7	I	umbilical hernia	Mesh repair	L1-L2	T11	105
28	MURUGAN	54/M	13577	23.7	II	B/lIng Hernia	Hernioplasty	L1-L2	T11	130
29	SIVACHIDHAMBARAM	56/F	57890	25.4	I	incisional hernia	Mesh repair	L1-L2	T11	140
30	RAVIRASU	57/M	24677	23.44	II	B/L Ing Hernia	Mesh repair	L1-L2	T11	110

SENSORY ONSET AT TIO IN MINS	MAX SENSORY BLOCK LEVEL	MAX SENSORY BLOCK LEVEL IN MINS	TIME FOR TWO SEGMENT LEVEL REGRESSION	ONSET OF MOTOR BLOCK IN MINS	TIME OF COMPLETE MOTOR BLOCK	TIME OF RESCUE ANALGESIA IN MINS	SIDE EFFECTS	
7	T4	12	90	10	14	150		
9	T4	14	70	9	15	155	dizziness	
10	T5	15	60	13	14	160	hypotension	
8	T4	16	80	12	16	165		
7	T4	14	90	10	14	160	drymouth, hypotension	
6	T4	12	100	12	15	160	brdy,hypoten	
7	T4	14	85	10	13	170	brady	
7	T4	12	90	9	11	180		
7	T4	12	90	12	15	170		
8	T5	14	100	14	16	160		
6	T4	15	80	10	14	150		
7	T4	16	70	13	16	160	hypotension	
8	T4	12	80	9	12	190		
7	T6	14	70	10	14	170	dry mouth	
6	T4	12	60	12	15	150		
7	T6	12	60	11	16	150	bradycardia	
8	T4	12	65	12	16	150		
7	T4	14	80	10	15	155	shivering	
6	T4	12	90	10	14	165		
6	T6	14	90	10	14	165	hypotension	
7	T4	15	95	14	16	145		
7	T4	16	90	10	14	140		
7	T4	14	90	10	15	135		
6	T4	12	95	10	15	150		
9	T4	12	100	10	14	165	shivering	
9	T4	14	80	10	14	185		
8	T5	15	90	9	13	165		
8	T4	16	100	8	15	139		
6	T4	15	90	10	14	140		
6	T4	14	80	12	14	145		

SYSTOLIC BP mmHg

DIASTOLIC BP mmHg

2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins
126	130	100	96	94	100	104	120	110	86	90	84	74	74	80	76	84	82
130	126	114	98	90	96	100	102	110	80	96	80	80	76	84	80	78	80
122	120	110	100	96	96	110	110	114	82	94	76	78	72	80	80	80	82
120	130	100	98	94	100	102	102	104	82	92	90	80	74	82	82	80	78
120	110	94	88	90	108	110	110	112	76	80	78	74	76	68	80	80	74
120	106	96	90	90	96	102	104	102	74	84	74	64	68	70	74	76	72
126	124	96	88	86	96	112	110	110	72	86	76	64	62	66	70	68	68
110	112	100	90	90	98	114	114	110	70	84	72	70	66	68	74	66	66
118	116	110	90	88	94	116	114	114	70	88	70	60	66	66	72	70	70
116	110	100	90	96	100	116	112	112	70	82	68	58	60	64	66	72	68
136	122	110	100	96	102	102	106	104	68	80	70	56	60	62	66	70	68
114	124	100	90	90	104	104	106	116	68	78	64	70	66	64	74	70	62
128	130	102	96	96	106	108	102	104	70	76	66	70	68	66	76	74	64
136	140	120	96	94	108	120	110	116	78	74	68	64	60	64	68	72	70
112	130	100	94	90	102	104	106	128	76	70	60	58	60	64	78	74	72
132	120	100	94	90	100	118	120	124	74	72	62	60	64	60	74	74	74
136	100	98	90	86	106	114	116	122	70	70	64	58	58	66	76	76	76
124	104	90	86	88	110	110	114	120	76	86	68	62	64	70	72	70	76
122	106	100	88	90	100	100	118	124	78	84	68	64	66	68	68	76	74
128	108	94	90	90	102	102	116	118	74	70	68	62	64	66	72	76	76
120	110	98	90	92	110	106	120	114	70	68	66	64	66	68	76	78	74
130	110	100	96	96	102	110	114	112	70	66	70	68	64	66	70	80	72
124	120	110	100	94	106	116	118	120	68	84	64	60	62	64	64	70	70
120	114	104	100	96	98	118	120	122	68	82	78	66	56	68	74	68	70
140	116	102	98	96	100	120	122	124	72	80	84	56	68	66	70	66	72
126	118	100	96	94	102	122	124	126	74	86	70	64	60	66	68	66	74
128	120	100	96	90	104	124	126	128	76	84	70	70	54	68	70	64	64
126	116	102	94	90	106	122	130	124	80	84	68	56	64	68	64	66	66
140	110	104	96	90	102	126	132	126	80	82	60	74	78	74	70	70	64
138	110	102	90	94	112	124	136	130	68	80	62	60	68	62	68	70	66

PULSE RATE

RESPIRATORY RATE

2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins
86	102	80	74	70	78	77	78	81	14	14	14	14	14	14	14	14	14
80	100	82	70	72	80	80	85	80	16	16	14	14	14	14	14	14	14
82	98	66	70	74	74	85	84	83	14	14	14	14	14	14	14	14	14
82	110	68	66	70	75	80	86	84	14	14	14	14	14	14	14	14	14
76	94	62	65	69	74	77	78	86	14	14	14	13	14	14	14	14	14
74	90	63	68	68	71	79	82	85	16	16	14	14	14	14	14	14	14
72	112	62	66	66	77	78	80	88	16	16	14	14	14	14	14	14	14
70	76	69	67	67	78	79	76	86	16	16	14	14	14	14	14	14	14
70	115	64	69	61	72	78	78	84	16	16	14	14	14	14	15	14	15
70	104	67	67	65	74	73	73	82	16	16	15	14	15	14	14	14	14
68	84	68	68	68	75	76	79	87	16	16	14	16	14	14	14	15	14
68	86	64	67	69	77	78	74	89	16	16	14	14	14	14	14	14	15
70	104	64	70	63	76	77	75	82	16	16	16	15	14	14	14	14	14
78	82	72	71	62	79	76	76	83	16	16	14	14	14	14	14	15	15
76	88	70	72	64	69	74	77	85	16	16	14	14	16	14	14	14	15
74	96	66	70	61	66	70	78	88	16	16	14	14	14	15	14	14	14
70	110	63	73	74	64	71	74	89	16	16	14	14	14	14	14	14	15
76	78	61	71	75	68	72	71	80	16	16	14	15	14	14	14	14	16
78	96	62	70	64	62	70	79	78	16	16	15	14	14	14	14	14	16
74	109	68	74	61	63	74	72	89	16	16	14	15	14	15	14	14	16
70	98	72	70	63	69	71	70	89	16	16	14	14	14	14	14	14	15
70	104	74	69	69	62	76	78	88	16	16	15	14	14	14	14	14	16
68	106	75	60	65	61	71	74	87	16	16	14	14	15	14	14	16	15
68	68	70	63	68	64	70	72	86	16	16	14	14	14	14	14	14	16
72	112	74	70	64	63	69	74	89	16	16	14	14	14	14	14	14	15
74	85	72	71	65	62	68	70	90	15	15	16	14	14	14	14	14	15
76	116	78	63	66	67	62	72	93	16	16	16	14	15	14	14	14	16
80	115	79	76	69	66	68	69	94	16	16	14	14	14	14	14	14	15
80	79	74	72	70	65	65	68	92	14	14	14	14	14	14	14	14	14
68	114	74	74	76	64	66	67	91	16	16	14	15	14	14	14	14	14

SpO2

SEDATION SCORE

2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins	2 Mins	5 Mins	10 Mins	15 Mins	20 Mins	25 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins
99	99	99	99	99	99	99	99	99	1	2	2	2	2	2	2	2	2	2	2
99	99	99	99	99	99	99	100	99	2	2	2	2	2	2	2	2	2	2	2
100	100	99	99	99	99	99	99	100	2	1	1	3	3	2	2	2	2	1	2
99	99	99	99	99	99	99	99	99	2	1	1	3	2	3	3	3	3	2	2
99	99	99	99	99	99	100	99	99	2	2	2	3	3	2	2	2	2	2	2
99	99	99	99	99	98	99	99	99	2	1	2	4	3	2	2	2	2	2	3
99	99	99	99	99	99	99	99	99	2	2	2	3	2	2	2	2	2	2	2
99	99	99	99	99	99	99	100	99	2	1	1	2	2	3	3	3	1	2	2
99	99	99	99	99	99	99	99	100	2	2	2	3	3	3	2	2	2	2	2
99	99	99	99	99	99	100	99	99	2	2	2	3	3	3	3	3	2	2	2
99	99	98	99	99	99	99	99	99	2	1	2	4	3	3	2	2	2	2	2
99	99	99	98	100	100	99	99	99	2	2	1	2	2	3	3	2	2	2	2
99	99	99	99	99	99	99	99	99	2	2	2	3	3	3	3	3	3	2	2
99	99	100	99	99	99	99	99	99	2	2	2	2	2	3	3	2	2	2	2
99	99	99	99	99	99	99	99	99	2	2	2	2	2	3	2	3	2	2	2
98	98	99	98	98	100	99	99	99	1	2	2	3	3	3	2	2	2	3	2
98	98	99	99	99	99	99	99	99	2	2	2	2	4	3	3	3	3	2	2
98	98	99	99	99	99	99	99	99	2	2	2	4	3	2	2	2	2	2	1
99	99	99	99	99	99	98	100	99	2	1	1	3	4	3	3	3	3	2	2
100	100	99	99	100	99	99	99	98	1	2	2	2	4	3	2	2	2	3	2
100	100	99	99	99	100	99	99	99	2	2	2	2	2	2	3	3	2	2	2
100	100	98	100	99	99	99	98	99	2	2	2	3	3	3	2	2	2	2	2
99	99	99	99	99	99	99	99	99	2	1	1	2	3	2	3	3	3	2	2
98	98	100	99	98	99	98	98	99	2	2	2	3	3	2	2	2	2	3	2
99	99	99	99	99	98	99	99	99	2	2	2	2	2	3	3	3	2	2	2
99	99	99	99	99	99	99	99	99	2	2	2	3	3	2	2	2	2	2	2
99	99	99	99	99	99	99	99	99	2	2	2	3	2	2	2	2	2	2	2
99	99	99	99	98	99	98	99	99	2	2	2	3	2	3	2	2	2	2	2
99	99	99	100	99	98	99	98	99	2	2	2	3	3	3	2	2	2	2	2
99	99	99	99	99	99	99	98	98	2	1	2	3	3	3	3	2	2	2	2

GROUP-ID

S. No.	NAME	AGE / SEX	IPNO	BMI	ASA	DIAGNOSIS	PROCUDURE	EPIDURAL SITE	CATHETER TIP	AVERAGE DURATION OF SURGERY IN MINS
1	RAJAPPA	45/M	34556	16.36	II	B/L ING HERNIA	Hernioplasty	L1-L2	T11	100
2	PANDIYAN	54/M	24454	20.09	I	B/L Ing Hernia	Hernioplasty	L1-L2	T11	115
3	PANDIAMMAL	47/F	24556	20	II	B/L ing hernia	Hernioplasty	L1-L2	T11	90
4	MANIKANDAN	63/M	86656	25.9	I	B/L ing hernia	Hernioplasty	L1-L2	T11	120
5	RAKKU	65/F	24545	25.25	I	B/L ingheria	Hernioplasty	L1-L2	T11	115
6	RAJESHWARI	49/F	87664	24.1	I	B/L ingheria	Hernioplasty	L1-L2	T11	120
7	KRISHNAN	48/M	35678	24.54	II	B/L ingheria	Hernioplasty	L1-L2	T11	130
8	MANIMARAN	48/M	75336	27.27	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	120
9	AROKYAMARY	45/F	96547	16.94	II	B/L ing hernia	Hernioplasty	L1-L2	T11	125
10	THNGAPPAN	46/M	97655	17.68	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	120
11	SUGANTHI	56/F	23445	21	II	umbilical hernia	Mesh repair	L1-L2	T11	100
12	KANDHAN	55/M	77689	21.33	II	U/L ing.hernia	Hernioplasty	L1-L2	T11	105
13	SANTHANAM	58/M	24346	19.29	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	120
14	KALAIVANI	57/F	33434	26.8	II	umbilical hernia	Hernioplasty	L1-L2	T11	60
15	RAJASEKARAN	52/M	97676	28.5	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	90
16	MOORTHY	45/M	67678	28.42	I	B/L ingheria	Hernioplasty	L1-L2	T11	100
17	DEIVANAI	63/F	34454	24.01	II	incisional hernia	Mesh repair	L1-L2	T11	100
18	NACHIAPPAN	61/M	97676	25.8	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	105
19	KALYANI	60/F	12233	36.11	II	incisional hernia	Mesh repair	L1-L2	T11	110
20	SANTHOSAM	60/M	34556	25	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	130
21	KARTHIKA	50/F	97876	34.73	I	incisional hernia	Mesh repair	L1-L2	T11	160
22	BASKAR	50/M	86676	28.98	I	U/L ing hernia	Hernioplasty	L1-L2	T11	110
23	VIRUMANDI	50/M	76454	27.82	II	UL ing hernia	Hernioplasty	L1-L2	T11	120
24	VIJAYALAKSHMI	52/F	86545	25.2	I	incisional hernia	Mesh repair	L1-L2	T11	140
25	MANI	51/F	54544	25.85	I	incisional hernia	Mesh repair	L1-L2	T11	140
26	NAGARAJ	49/M	75654	26.11	II	U/L ing.hernia	Hernioplasty	L1-L2	T11	90
27	MUNUSAMY	53/M	54433	21.42	I	B/L ingheria	Hernioplasty	L1-L2	T11	105
28	POONGATHAI	58/F	34343	20.86	I	incisional hernia	Mesh repair	L1-L2	T11	130
29	PAZHANI	57/M	97876	23.1	I	U/L ing.hernia	Hernioplasty	L1-L2	T11	150
30	SAKTHI	58/F	64545	22.65	I	incisional hernia	Mesh repair	L1-L2	T11	110

SENSORY ONSET AT TIO IN MINS	MAX SENSORY BLOCK LEVEL	MAX SENSORY BLOCK LEVEL IN MINS	TIME FOR TWO SEGMENT LEVEL REGRESSION	ONSET OF MOTOR BLOCK IN MINS	TIME OF COMPLETE MOTOR BLOCK	TIME OF RESCUE ANALGESIA IN MINS	SIDE EFFECTS	
10	T4	14	35	8	10	60		
11	T4	15	40	10	14	65	hypotension	
15	T5	18	45	9	14	70		
10	T4	14	50	12	15	65	brady,hypotension	
11	T4	15	45	10	15	60	dry mouth	
13	T4	16	40	10	15	80		
14	T4	18	40	10	15	105	dry mouth	
14	T4	18	45	9	12	60	bradycardia	
14	T4	18	45	8	12	70		
12	T5	16	50	9	12	80		
15	T4	19	35	10	15	90	hypotension	
15	T4	18	40	10	15	60	hypotension	
15	T4	18	40	12	16	60		
14	T6	18	40	12	16	60	dry mouth	
12	T4	16	45	8	12	65	bradycardia	
14	T6	18	45	9	12	70		
14	T4	18	40	10	15	70		
10	T4	15	45	10	15	75		
15	T4	18	45	10	15	80	bradycardia	
11	T6	15	45	12	16	85	bradycardia	
12	T4	15	50	15	18	75	drymouth	
12	T4	15	50	13	16	70		
14	T4	18	50	12	16	80		
12	T4	16	45	12	16	70		
15	T4	18	40	9	12	60		
10	T4	15	40	10	15	60		
12	T5	15	40	8	12	60	brady,hypotens	
10	T4	15	40	9	15	65	drymouth	
12	T4	16	40	10	14	60	drymouth	
12	T4	16	45	9	12	60		

SYSTOLIC BP

DIASTOLIC BP

2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins
130	94	90	100	102	118	120	120	122	72	58	60	60	64	66	64	70	70
120	94	90	100	100	114	116	122	132	70	60	62	62	56	60	72	70	78
100	90	86	98	106	110	114	116	136	86	58	58	64	66	70	74	72	76
104	88	88	90	110	100	118	120	124	84	60	64	68	68	74	74	74	74
106	88	90	100	100	102	116	120	122	70	64	66	68	66	76	76	76	70
108	90	90	94	102	106	120	124	128	68	66	64	68	64	72	70	76	76
110	90	92	98	110	110	114	112	120	66	64	66	70	62	68	76	74	78
110	96	96	100	102	116	118	120	130	84	68	60	72	60	72	76	76	76
120	100	94	110	106	118	120	122	124	82	60	62	64	60	74	78	74	70
114	98	96	104	106	120	122	126	126	80	66	56	78	64	70	72	80	80
116	98	96	102	100	122	124	126	140	86	70	68	84	70	64	70	74	68
118	102	94	100	102	124	126	128	126	84	70	60	82	72	74	68	70	70
120	96	90	100	104	122	130	134	130	84	70	54	80	68	70	66	72	72
116	94	90	102	106	126	132	132	130	82	70	64	68	62	68	66	74	74
110	96	90	104	106	124	136	138	140	80	74	56	66	70	70	64	66	76
110	90	94	102	112	104	120	110	126	90	70	68	70	72	64	66	66	80
126	96	94	100	100	100	102	110	128	96	80	74	72	70	72	70	64	80
120	98	90	114	116	110	110	114	122	94	84	76	80	84	80	70	82	88
130	100	96	110	112	102	102	104	124	92	82	72	86	88	80	80	78	86
110	98	94	100	100	110	110	112	116	80	80	74	90	62	66	78	82	80
106	90	88	94	108	102	104	104	114	84	74	76	72	70	80	80	84	82
124	90	90	96	96	112	110	110	116	86	66	68	74	74	80	80	80	82
112	86	86	96	96	114	114	112	114	84	64	62	70	70	70	68	72	74
116	90	90	100	102	116	114	116	118	88	70	66	72	74	74	76	68	76
110	90	88	110	112	116	112	112	118	82	60	58	70	72	68	68	66	74
122	90	96	100	100	102	106	108	116	80	58	60	68	66	68	66	70	70
124	102	96	110	110	104	106	116	126	78	56	60	64	66	68	70	68	72
130	88	86	100	104	108	102	104	114	76	72	66	70	68	66	72	74	76
140	96	96	102	106	120	110	116	128	74	70	68	66	70	70	70	76	78
126	96	94	120	116	104	106	128	136	70	68	60	68	66	74	70	78	66

PULSE RATE

RESPIRATORY RATE

2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins	2 Mins	5 Mins	10 Mins	15 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins
112	74	70	66	75	76	76	87	82	14	14	14	15	14	14	14	14	14
100	68	66	64	69	70	75	84	85	14	14	14	14	14	14	14	14	14
105	66	62	54	66	71	75	82	84	14	14	14	14	14	14	14	14	14
110	68	66	74	64	68	74	83	86	14	14	14	14	14	14	14	14	15
106	58	66	70	70	70	74	85	81	14	14	12	16	14	14	14	14	15
100	63	71	66	64	70	71	78	78	15	14	14	15	14	14	15	14	15
112	62	64	66	63	66	79	81	75	14	12	14	15	12	14	15	14	16
78	60	66	63	69	76	72	80	79	14	14	14	15	14	14	15	14	16
110	64	68	70	62	71	70	78	85	14	14	14	14	14	14	15	14	16
108	67	69	65	61	65	70	89	85	14	14	14	14	14	14	15	14	16
84	68	60	68	66	69	74	76	87	14	14	14	14	14	12	15	15	19
74	64	63	66	63	68	72	78	84	14	14	14	16	15	14	15	14	18
104	64	68	65	62	62	74	84	88	14	15	12	14	16	14	14	14	15
82	60	71	70	67	68	70	86	85	14	12	14	16	16	14	16	15	14
98	70	63	58	66	65	72	89	86	14	14	14	14	16	14	16	14	14
96	60	70	70	65	66	69	90	89	14	14	14	15	15	14	16	16	15
110	63	66	72	64	68	68	78	80	14	12	14	16	15	14	16	14	16
78	59	70	71	78	79	80	94	97	14	14	14	14	15	14	15	18	14
96	58	66	72	76	80	78	86	85	15	14	14	14	15	14	14	14	15
109	64	70	74	76	80	85	86	88	14	14	14	14	15	14	15	18	18
98	70	64	70	74	77	79	81	85	14	14	14	14	15	14	16	14	15
104	72	66	69	74	76	80	84	86	14	14	14	15	15	15	14	16	16
106	70	65	68	70	78	78	83	84	14	12	14	15	14	14	15	14	15
70	64	68	66	69	70	82	84	85	14	14	14	15	14	14	16	15	14
112	66	66	67	70	74	80	86	90	14	14	14	15	14	14	16	14	15
88	72	67	60	72	73	74	80	84	14	14	14	16	14	14	15	16	14
110	78	69	64	70	74	78	84	88	14	14	14	15	14	14	14	16	14
112	74	67	66	69	70	73	76	77	14	12	14	14	14	14	12	14	15
85	66	60	65	73	75	76	84	89	14	14	12	14	12	14	5	15	15
112	80	67	60	72	75	75	80	83	14	14	14	14	14	14	14	14	16

SpO2

SEDATION SCORE

2 Min	5 Mins	10 Min	15 Min	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins	2 Min	5 Mins	10 Min	15 Mins	20 Mins	25 Mins	30 Mins	45 Mins	60 Mins	90 Mins	120 Mins
99	99	99	99	99	99	99	99	98	2	2	3	3	3	2	2	2	2	2	1
99	99	99	99	99	99	99	99	100	1	2	3	2	2	2	2	2	2	2	1
99	99	100	98	99	99	99	99	100	2	2	4	4	2	2	2	2	2	2	2
99	99	99	99	100	98	98	98	99	2	2	3	3	3	3	3	3	2	2	1
99	99	99	98	99	99	99	99	99	1	2	3	3	2	2	2	2	2	2	2
99	100	99	99	99	99	100	99	100	2	2	4	4	4	3	2	2	2	2	2
99	100	100	99	99	99	100	99	99	2	2	3	3	4	2	2	2	2	2	2
99	99	99	99	99	99	99	99	99	1	2	2	2	3	3	3	3	2	2	2
99	99	99	99	99	100	99	100	99	2	2	3	3	3	3	3	3	2	2	2
99	100	99	100	98	99	99	99	100	2	2	3	3	3	3	3	2	3	2	2
100	98	100	100	99	99	99	99	100	1	2	4	4	3	2	2	2	2	2	2
99	100	100	99	99	98	100	99	99	2	3	3	3	2	3	3	2	2	2	2
99	99	98	99	99	98	98	98	99	2	3	3	3	3	3	3	3	2	2	2
99	99	98	99	99	99	99	99	100	2	2	2	4	4	4	3	3	2	2	2
99	100	100	100	99	98	99	99	98	2	2	2	3	3	2	3	2	2	3	2
98	99	99	100	100	99	99	99	100	2	2	3	3	3	2	2	2	2	2	2
99	98	100	100	99	98	100	99	100	2	2	3	4	3	3	3	3	3	2	2
99	100	99	100	99	99	99	100	99	2	2	3	3	3	3	2	2	2	2	2
99	99	98	99	99	99	99	99	100	2	2	4	3	3	2	2	2	2	2	2
100	99	99	99	99	98	99	99	100	2	2	3	4	3	2	2	2	2	2	2
99	99	99	99	98	99	99	99	99	2	2	3	2	2	3	3	3	3	2	2
99	100	100	99	99	98	98	99	100	2	2	2	3	3	3	2	2	2	2	2
99	100	100	99	99	99	99	99	99	1	2	4	4	2	3	3	3	3	2	2
98	99	100	99	99	99	99	98	100	2	2	3	2	3	2	2	2	2	2	2
99	100	100	98	98	100	100	99	100	1	2	4	4	4	3	3	3	3	2	2
99	99	99	98	99	99	99	99	99	1	2	3	3	3	2	2	2	2	3	3
99	99	98	100	99	99	99	99	100	1	2	3	3	4	3	2	2	3	2	2
99	100	99	99	99	99	100	100	100	2	3	4	3	3	2	2	2	2	2	2
99	100	100	100	99	99	99	99	100	1	2	3	3	3	2	2	2	2	2	2
99	99	98	100	99	98	100	99	100	2	2	3	4	3	3	3	3	3	2	2

COMPARATIVE STUDY TO ASSESS THE EFFICACY OF EPIDURAL DEXMEDETOMIDINE AND INTRAVENOUS DEXMEDETOMIDINE IN PATIENTS UNDERGOING LOWER ABDOMINAL SURGERIES UNDER EPIDURAL ANAESTHESIA

DISSERTATION SUBMITTED FOR THE DEGREE OF

DOCTOR OF MEDICINE

BRANCH – X (ANAESTHESIOLOGY)

APRIL-2013

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

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